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Movement Disorders Associated with Moyamoya Disease: A Report of 4 New Cases and a Review of Literatures

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Abstract: The aim of this study was to define the clinical characteristics of patients who developed movement disorders in association with moyamoya disease (MMD). Using PubMed and medical records of our hospital from 1985 to 2008, we searched for patients who developed movement disorders in association with MMD. This study included 38 patients described in previous studies and 4 patients found in the medical records. The onset of movement disorders was thought to be sudden. In 13 patients, the movement disorders were precipitated by hyperventilation or emotional stress. Twenty-seven of the 42 patients developed chorea, 4 patients developed dystonia, and 4 developed a mixture of both. The movement disorders of the remaining 7 patients were described as dyskinesia. A third of the 42 patients developed bilateral movement disorders, and their mean age was younger than that of those with unilateral movement disorders. In 37 of the 42 patients, brain imaging studies showed ischemic lesions, but the remaining 5 patients showed no parenchymal lesions. Cerebral perfusion studies showed hypoperfusion in the basal ganglia and in the cerebral cortical areas. Most patients improved whether they were treated or not. MMD must be included in the differential diagnosis of the sudden onset of dyskinesias, particularly chorea and focal dystonia. Even in patients with no parenchymal lesion in brain imaging studies, cerebral angiography and cerebral blood perfusion studies must be performed, if they develop a sudden onset or recurrent movement disorders preceded by emotional stress or hyperventilation. © 2010 Movement Disorder Society

Key words: movement disorders; moyamoya disease; chorea; dystonia; dyskinesia

Moyamoya disease (MMD) is characterized by progressive occlusion of the internal carotid artery (ICA) and the main branches within the circle of Willis. It frequently manifests as transient ischemic attack or seizure in children, but hemorrhagic stroke is also common in adults.[1] MMD occurs frequently in females and Asians.

Up to 6% of patients with MMD may develop various movement disorders, including chorea, dystonia, and dyskinesia, as an initial manifestation or during their clinical courses.[2,3] However, such patients were reported as either a stand-alone case or in a small series.[2–28] As a result, the overall clinical features of movement disorders associated with MMD are not well defined. We analyzed demographic characteristics, clinical features, and radiological findings of 4 patients from our hospital and 38 reported patients who developed movement disorders in association with MMD.

METHODS

Using PubMed from 1985 to 2008, we carried out multiple searches with nine key words (e.g., moyamoya, dyskinesia, tremor, dystonia, chorea, athetosis, myoclonus, seizure, and convulsion). In addition to full original articles, cases reported in abstract form were also included, if detailed clinical findings were described. Regarding the terminology for movement disorders, we have quoted the authors' description. We also searched the medical record of Gangnam Severance Hospital, Yonsei University College of Medicine, in the same time period find patients who developed movement disorders in association with MMD. We an-

Additional Supporting Information may be found in the online version of this article.

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alyzed demographic characteristics, predisposing factors, modes of onset, affected body parts, neuroradiological findings, treatments, and clinical courses of movement disorders associated with MMD. Informed written consent was not obtained, and ethical approval was waived because this study did not require of those.

RESULTS

We found 38 patients in 27 studies (Supporting Information Table 1),[2–28] including 2 patients we previously reported.[10,26] We also found 4 new patients in our medical records from 1985 to 2008.

Demographics and Clinical Characteristics of 42 Patients Who Developed Movement Disorders in Association With MMD

Thirty-two of the 42 patients (76.2%) were Asian. Thirty-one (73.8%) patients were female and 11 (26.2%) were male (Table 1). The mean age at the onset of the movement disorders was 21.4 years (range = 1–61 years). Family histories were mentioned in 15 patients. Three (20%) of them had other family members with MMD. However, none of the family members had a history of movement disorders.

Among the 42 patients included in this study, 27 (64.3%) had chorea, 4 (9.5%) had dystonia, and 4 (9.5%) had both. The remaining 7 patients (16.7%) were described to have dyskinesia. In all 42 patients, the onset was sudden. Movement disorders were transient in 15 (35.7%) of the 42 patients: 5 with chorea, 2 with dystonia, 1 with chorea and dystonia, and 7 with dyskinesia. Movement disorders occurred recurrently in 15 of the 42 patients. In 13 of them, movement disorders were predisposed by various conditions, inducing hyperventilation or emotional stress (e.g., singing, blowing, eating hot and spicy food, jogging, walking up the stairs, swimming, crying, sexual coitus, and job insecurity).

Twenty-eight of the 42 patients (66.7%) developed movement disorders affecting one side of the body and the remaining 14 patients (33.3%) developed bilateral movement disorders. The mean age at the onset of the unilateral movement disorders (13.3 ± 7.3 years) was younger than that of the bilateral movement disorders (25.2 ± 16.4 years; $P < 0.005$). Eleven of the 14 patients with bilateral symptoms had chorea, 2 had both chorea and dystonia, and 1 had dyskinesia. However, none of them had bilateral dystonia. Among the 4 patients with pure dystonia, 1 patient developed hemidystonia and the remaining 3 developed focal dystonia affecting a leg, an arm, or neck. However, none of them had facial dystonia. In 1 patient, pain was accompanied in the hand affected by dystonia. None had a sensory trick.

TABLE 1. *Demographic and clinical characteristics of MD-MMD*

Total patients	42
Women	31 (73.8%)
Men	11 (26.2%)
Asian	32 (76.2%)
Non-Asian	10 (23.8%)
Mean age at onset of MD	21.4 yr (1–62)
Mean duration for follow up	18.9 mo (3-48)
MD symptom[a]	
Chorea	31 (67.4%)
Arm only	3 (9.7%)
Arm and leg	18 (58.1%)
Arm and face	2 (6.5%)
Arm, leg and face	8 (25.7%)
Dystonia	8 (17.4%)
Hand	2 (25%)
Foot	2 (25%)
Arm and leg	2 (25%)
neck	2 (25%)
Dyskinesia	7 (15.2%)
arm	5 (71.4%)
arm and leg	2 (28.6%)
Mode of symptom onset	
Constant	27 (64.3%)
Paroxysmal	15 (35.7%)
Laterality	
Bilateral	14 (33.3%)
Unilateral	28 (66.7%)
Treatment	30
Medication	6 (20.0%)
Surgery	21 (70.0%)
both	3 (10.0%)
No treatment	10
Prognosis	40
Subside	32 (80%)
Improved	4 (10%)
Persist	4 (10%)

MD, movement disorders; MMD, moyamoya disease.
[a]Including four patients who have overlap symptoms with both chorea and dystonia.

Six of the 27 patients with isolated chorea also had iatrogenic or primary endocrine dysfunction. Three patients developed chorea, while they were taking oral contraceptives.[8,17] In all 3 patients, chorea disappeared within a week after the discontinuation of oral contraceptives. Two patients developed chorea while they were pregnant.[12,19] One of them improved after a therapeutic abortion.[12] The remaining patient had Graves' disease and improved after thyroidectomy.[27] In 7 of the 42 patients, the term "dyskinesia" was used for the description of the movement disorders.[19–21,26] All 7 patients suddenly developed dyskinesia repeatedly. Four of those 7 patients developed unilateral limb shaking, which lasted for a very short period of time, as observed in limb shaking TIAs.

Thirty of the 40 patients, in whom the treatment methods were described, received medical treatment or underwent vascular surgery. In 8 patients with bilateral movement disorders, the results of the vascular bypass surgery were described. Most patients improved after surgical treatment.[2,3,11,21,24,28] However, 10 patients received no specific treatment. The mean duration of the follow-up period was 18.9 months (range = 3–48 months). Among 30 patients who were treated, 28 of them either completely or partially improved, but 2 patients (one treated with vascular surgery and the other with medication) did not respond at all. On the other hand, 8 of the 10 patients who did not receive any treatment improved for their own accord. Four of them improved spontaneously a week to 15 months after the onset of movement disorders. The remaining 4 improved after the discontinuation of oral contraceptives or therapeutic abortion.

Neuroradiological Findings of Patients With Movement Disorders Associated With MMD

In all 42 patients, the diagnosis of MMD was confirmed by conventional or magnetic resonance (MR) angiography studies. (Table 2). Forty (95.2%) patients had lesions in the ICA, and the remaining 2 patients (4.8%) had occlusion or stenosis in the middle cerebral artery (MCA). Most patients (85.7%) had occlusion or stenosis in the bilateral ICA or MCA. Occlusion (66.7%) was more frequent than stenosis (33.3%).

Brain computerized tomography (CT) or magnetic resonance imaging (MRI) studies performed after the onset of movement disorders showed an ischemic lesion in 36 (86%) patients. Lesions were frequently found in the subcortical area (48.2%; 26 among 54 lesions), including the periventricular white matter or centrum semiovale, and the basal ganglia (26%; 14 among 54 lesions). One patient showed a hemorrhage in the basal ganglia contralateral to the hand affected by dystonia (Present Case 1).

However, the remaining 5 (12%) patients showed no parenchymal lesions. Four of the 5 patients presented with chorea (see Case 4 in this article),[13,16,28] and the remaining patient presented with paroxysmal exercise induced dyskinesia.[26] Among these 5 patients, the all 4 patients, who underwent cerebral perfusion studies, showed reduced cerebral blood perfusion in the basal ganglia and cerebral cortex (see Case 4 in this article).[16,26,28]

All 24 patients who underwent cerebral perfusion studies were found to have reduced cerebral perfusion. Ten of them showed hypoperfusion outside of the basal ganglia or thalamus, and the remaining 14 patients showed hypoperfusion in the unilateral or bilateral basal ganglia or thalamus. Among these 14 patients, 11 patients showed no lesion in the basal ganglia or thalamus on brain CT or MRI studies.

TABLE 2. *Neuroimaging findings in MD-MMD*

CT or MRI	42
Ischemia	36 (85.7%)
Hemorrhage	0
Ischemia and hemorrhage	1 (2.4%)
None	5 (11.9%)
Ischemic lesions[a]	54
Cortex	13 (24.1%)
Subcortex	26 (48.2%)
BG	14 (25.9%)
Thalamus	1 (1.9%)
Ischemic cortical or subcortical area[a]	39 (72.3%)
Frontal	19 (48.7%)
Temporal	4 (10.3%)
Parietal	8 (20.5%)
Occipital	8 (20.5%)
Angiography	42
Bilateral ICA occlusion	22 (52.4%)
Bilateral ICA stenosis	13 (30.9%)
Bilateal MCA occlusion	1 (2.4%)
Unilateral ICA occlusion	5 (11.9%)
Unilateral ICA stenosis	1 (2.4%)
SPECT or PET	24
Lesions[a]	30
Cortex	13 (43.3%)
Subcortex	2 (6.7%)
BG	13 (43.3%)
Thalamus	2 (6.7%)

BG, Basal ganglia; ICA, internal carotid artery; MCA, middle cerebral artery.
[a]Anatomically, multiple lesion can be observed.

DISCUSSION

MMD affects females much more frequently than males, and the reported female to male ratio was 1.8.[29] In this study, the number of female patients was 2.5 times than that of male patients. The incidence of MMD is high in Asian countries. In Japan, the annual prevalence of MMD is 10.5 and the incidence is 0.94 per 100,000 individuals.[29] In this study, the number of Asian patients was three times higher than that of non-Asian patients.

Familial occurrence was found in 15% of patients with MMD.[29] A similar portion (20%) of patients included in this study had a positive family history of MMD. Nanba et al.[30] studied 24 familial MMD patients from 10 families. Their mean (±SD) age at the symptom onset (11.8 ± 11.7 years) was younger than that of nonfamilial cases (30.0 ± 20.9 years). Among 24 patients, 8 inherited MMD from their parents, and all were mother-offspring pairs. Their mean age at the onset was older than that reported by

Nanba et al.[30] None of the 3 familial MMD patients had family members who developed movement disorders in association with MMD. Therefore, familial chorea seems to rarely occur in MMD.

In this study, chorea was the most frequent movement disorder observed in MMD. Sixteen patients developed hemichorea and 11 developed generalized chorea. The most frequent cause of the sudden onset of hemichorea may be a lacunar infarction or neoplasm in the contralateral basal ganglia or subthalamic region.[31] In addition, hemibody is involved in about 90% of chorea associated with nonketotic hyperglycemia and in 20% of patients with Sydenham's chorea.[32] In patients with MMD, dilated moyamoya collateral vessels compress the basal ganglia or cause local ischemia and may lead to choreiform dyskinesia.[33]

In this study, 6 MMD patients developed chorea in association with altered endocrine function, including oral contraceptive medication,[8,17] pregnancy,[12,19] or thyroid disease.[27] Five of them were found with basal ganglia lesions on a brain MRI or single photon emission computed tomography studies. It has been suggested that sexual or thyroid hormones may cause basal ganglia dopaminergic dysfunction and striatal catecholamine receptor hypersensitivity.[32] Therefore, we suspect that basal ganglia hyoperfusion or an infarction caused by MMD may uncover the basal ganglia dysfunction associated with hormonal imbalance.

Patients with a lesion in the basal ganglia, particularly in the putamen, or thalamus frequently develop hemidystonia.[31,34,35] In this study, 4 of the 8 patients who developed isolated dystonia or both dystonia and chorea had basal ganglia lesions and 1 had a thalamic lesion. Most patients with dystonia following a basal ganglia or thalamic lesion initially develop hemiparesis and subsequently develop dystonia as the hemiparesis resolves over a period ranging from a few weeks to years.[31,34,35] The delay between the acute insult and dystonia may reflect the time needed for the recovery of motor output pathways and development of pathological circuitry.[36] Interestingly, all 4 patients in this study suddenly developed dystonia.

A focal dystonia following a stroke is uncommon, but if it occurs, it frequently affects the hand.[36] In this study, the hand was affected in 2 of the 6 patients who developed focal dystonia or both dystonia and chorea. LeDoux and Brady[37] reported 25 patients with secondary cervical dystonia associated with structural lesions in the central nervous system. Lesions frequently involved the brainstem and cerebellum. Therefore, they suggested that dysfunction of cerebellar afferent pathways plays an important role in the development of cervical dystonia. This study included 2 patients who developed cervical dystonia in association with MMD. One had a lesion in the internal capsule nearby the caudate nucleus,[6] and the other had a lesion in the frontal subcortical area.[21]

In this study, 15 patients had transient recurrent movement disorders, and in many of them, the movement disorders were precipitated by emotional stress, exercise, hyperventilation, or smoking. Such clinical features may resemble epilepsy. However, they had no epileptiform activities on ictal electroencephalography study, definite tonic-clonic jerking, typical Jacksonian march, eye-head turning, or evolution to a generalized seizure (see Case 4 in this article).[4,9,14,19–22] Such patients can also be misdiagnosed to have functional dyskinesia. One of the 15 patients who developed chorea had no parenchymal lesion in the brain MRI studies, but cerebral perfusion studies showed hypoperfusion in both temporoparietal areas (present Case 4). MMD should be added to the differential diagnoses of secondary paroxysmal movement disorders, even in individuals with preceding emotional stress and no parenchymal lesions in brain imaging studies. Patients with TIA may also present brief recurrent myoclonus, athetoid movement, or dystonic posturing. They have stenosis in the carotid artery and the dyskinesia is frequently precipitated by physical exercise.[38] We suspect that the sudden onset of transient and recurrent movement disorders observed in MMD may share a common pathogenesis and phenomenology with limb shaking transient ischemic attack. Ischemic damage to the neurons may increase membrane instability, excitatory neurotransmitter release, neuronal excitability, and consequent movement disorders.[39]

In MMD, surgical revascularization is known to be the most useful strategy for the normalization of cerebral hemodynamics and prevention of recurrences.[40] In this study, most (85.7%) patients had bilateral MMD, but 6 (14.3%) patients had unilateral MMD. In about a third of patients with unilateral MMD, the contralateral side may be affected within 5 years, especially in Asians and familial patients.[41] Therefore, as long-term follow-up study is needed to evaluate the beneficial effect of unilateral surgical revascularization. However, in this study, patients with bilateral MMD or bilateral movement disorders improved after unilateral vascular bypass surgery, possibly by the increased collateral blood flow though the circle of Willis.[2,3,11,21,24,28] However, 80% of patients who had no specific treatment for MMD also improved spontaneously or after a removal of the causative factors. Further long-term studies are needed to define the effectiveness of surgical or other medical treatments for the management and prevention of movement disorders associated with MMD.

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REFERENCES

1. Suzuki J, Kodama N. Moyamoya disease—a review. Stroke 1983;14:104–109.
2. Watanabe K, Negoro T, Maehara M, Takahashi I, Nomura K, Miura K. Moyamoya disease presenting with chorea. Pediatr Neurol 1990;6:40–42.
3. Pavlakis SG, Schneider S, Black K, Gould RJ. Steroid-responsive chorea in moyamoya disease. Mov Disord 1991;6:347–349.
4. Kim DS, Ahn MY, Lee GH, Lee BI. Moyamoya disease presenting with chorea. J Korean Neurol Assoc 1993;11:619–621.
5. Takanashi J, Sugita K, Honda A, Niimi H. Moyamoya syndrome in a patient with Down syndrome presenting with chorea. Pediatr Neurol 1993;9:396–398.
6. Yasutomo K, Hashimoto T, Miyazaki M, Kuroda Y. Recurrent torticollis as a presentation of moyamoya disease. J Child Neurol 1993;8:187–188.
7. Hosoya M. Moyamoya disease presenting initially with mental disturbance. No To Hattatsu 1997;29:471–475.
8. Pelletier J, Cabanot C, Levrier O, Thuillier JN, Cherif AA. Angiodysplasia of moyamoya type disclosed by choreic unvoluntary abnormal movements during oral contraception. Apropos of 2 cases. Rev Neurol 1997;153:393–397.
9. Han SH, Kim YG, Cha SH, Chung SY. Moyamoya disease presenting with singing induced chorea. J Neurol Neurosurg Psychiatry 2000;69:832–838.
10. Lyoo CH, Oh SH, Joo JY, Chung TS, Lee MS. Hemidystonia and hemichoreoathetosis as an initial manifestation of moyamoya disease. Arch Neurol 2000;57:1510–1512.
11. Parmar RC, Bavdekar SB, Muranjan MN, Limaye U. Chorea: an unusual presenting feature in pediatric moyamoya disease. Indian Pediatr 2000;37:1005–1009.
12. Unno S, Iijima M, Osawa M, Uchiyama S, Iwata M. A case of chorea gravidarum with moyamoya disease. Rinsho Shinkeigaku 2000;40:378–382.
13. Shanahan P, Hutchinson M, Bohan A, Donoghue DO, Sheahan K, Owens A. Hemichorea, moyamoya, and ulcerative colitis. Mov Disord 2001;16:570–572.
14. Bakdash T, Cohen AR, Hempel JM, Hoagland J, Newman AJ. Moyamoya, dystonia during hyperventilation and antiphospholipid antibody. Pediatr Neurol 2002;26:157–160.
15. Hong YH, Ahn TB, Oh CW, Jeon BS. Hemichorea as an initial manifestation of moyamoya disease: reversible striatal hypoperfusion demonstrated on single photon emission computed tomography. Mov Disord 2002;17:1380–1383.
16. Miura T, Kobayashi M, Sonoo M, Isii K, Shimizu T. An adult case of moyamoya disease presenting with transient hemichorea. Rinsho Shinkeigaku 2002;42:45–47.
17. Rezak M, Vergenz SM. Chorea in a 21-year-old female associated with multiple risk factors. Mov Disord 2002;17 (Suppl 5):S317.
18. Shprecher DR, Renner DR, Skalabrin EJ. Moyamoya associated hemichorea/hemiballism culminating in infarction. Mov Disord 2006;21 (Suppl 15):S354.
19. Gonzalez-Alegre P, Ammache Z, Davis PH, Rodnizky RL. Moyamoya-induced paroxysmal dyskinesia. Mov Disord 2003;18:1051–1056.
20. Kim HY, Chung CS, Lee J, Han DH, Lee KH. Hyperventilation-induced limb shaking TIA in moyamoya disease. Neurology 2003;60:137–139.
21. Im SH, Oh CW, Kwon OK, Cho BK, Chung YS, Han DH. Involuntary movement induced by cerebral ischemia: pathogenesis and surgical outcome. J Neurosurg 2004;100:877–882.
22. Spengos K, Tsivgoulis G, Toulas P, Vemmos K, Vassilopoulos D, Spengos M. Hyperventilation-enhanced chorea as a transient ischemic phenomenon in a patient with moyamoya disease. Eur Neurol 2004;51:172–175.
23. Kim YO, Kim TS, Woo YJ, Kim CJ, Oh CK. Moyamoya disease-induced hemichorea corrected by indirect bypass surgery. Pediatr Int 2006;48:504–506.
24. Zheng W, Wanibuchi M, Onda T, et al. A case of moyamoya disease presenting with chorea. Childs Nerv Syst 2006;22:274–278.
25. Li JY, Lai PH, Peng NJ. Moyamoya disease presenting with hemichoreoathetosis and hemidystonia. Mov Disord 2007;22:1983–1984.
26. Lyoo CH, Kim DJ, Chang H, Lee MS. Moyamoya disease presenting with paroxysmal exercise-induced dyskinesia. Parkinsonism Relat Disord 2007;13:446–448.
27. Garcin B, Louissaint T, Hosseini H, Blanc R, Fenelon G. Reversible chorea in association with Graves' disease and moyamoya syndrome. Mov Disord 2008;23:620–622.
28. Kamijo K, Matsui T. Dramatic disappearance of moyamoya disease-induced chorea after indirect bypass surgery. Neurol Med Chir 2008;48:390–393.
29. Kuroda S, Houkin K. Moyamoya disease: current concepts and future perspectives. Lancet Neurol 2008;7:1056–1066.
30. Nanba R, Kuroda S, Tada M, Ishikawa T, Houkin K, Iwasaki Y. Clinical features of familial moyamoya disease. Childs Nerv Syst 2006;22:258–262.
31. Lee MS, Marsden CD. Movement disorders following lesions of the thalamus or subthalamic region. Mov Disord 1994;9:493–507.
32. Cardoso F, Seppi K, Mair KJ, Wenning GK, Poewe W. Seminar on choreas. Lancet Neurol 2006;5:589–602.
33. Scott RM, Smith JL, Robertson RL, Madsen JR, Soriano SG, Rockoff MA. Long-term outcome in children with moyamoya syndrome after cranial revascularization by pial syngiosis. J Neurosurg (Pediatr 2) 2004:100;142–149.
34. Bhatia KP, Marsden CD. The behavioural and motor consequences of focal lesions of the basal ganglia in man. Brain 1994;117:859–876.
35. Chuang C, Fahn S, Frucht SJ. The natural history and treatment of acquired hemidystonia: report of 33 cases and review of the literature. J Neurol Neurosurg Psychiatry 2002;72:59–67.
36. Kim JS. Delayed onset mixed involuntary movements after thalamic stroke: clinical, radiological and pathophysiological findings. Brain 2001;124:299–309.
37. LeDoux MS, Brady KA. Secondary cervical dystonia associated with structural lesions of the central nervous system. Mov Disord 2003;18:60–69.
38. Yanagihara T, Piepgras DG, Klass DW. Repetitive involuntary movement associated with episodic ischemia. Ann Neurol 1985;18:244–250.
39. Alberti A, Paciaroni M, Caso V, Venti M, Palmerini F, Agnelli G. Early seizures in patients with acute stroke: frequency, predictive factors, and effect on clinical outcome. Vasc Health Risk Manag 2008;4:715–720.
40. Smith ER, Scott RM. Progression of disease in unilateral moyamoya syndrome. Neurosurg Focus 2008;24:E17.
41. Hirotsune N, Meguro T, Kawada S, Nakashima H, Ohmoto T. Long-term follow-up study of patients with unilateral moyamoya disease. Clin Neurol Neurosurg 1997;99 (suppl 2):S178–S181.