Multiple sclerosis and parkinsonism: a case report

Vesile Ozturk
Egemen İdiman
Ihsan S. Sengun
Zafer Yuksel*

Department of Neurology,
*Department of Neurosurgery,
Faculty of Medicine,
Dokuz Eylül University,
Izmir, Turkey

Reprint requests to: Dr Vesile Ozturk,
Dokuz Eylül University, Medical Faculty,
Department of Neurology,
35340, Inciralti, Izmir, TURKEY
E-Mail: ozturkv@hotmail.com

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Summary

Multiple sclerosis (MS) is a well-known disease characterized by the distribution of plaques in the periventricular and subcortical white matter. Although plaques can also be found in the striatum, pallidum and thalamus, extrapyramidal symptoms are very rare in MS. However, the association of MS and parkinsonism is still a controversial topic as it has not been established whether these two conditions occur coincidentally or causally. In the literature, eleven cases of parkinsonism associated with MS have been described. Here, we report a patient with clinically definite MS and signs of parkinsonism. Our patient had slow progressive bradykinesia, static tremor and bradydysmia that were not associated with exacerbation or progression of the MS. This rare and interesting association of multiple sclerosis with parkinsonism is discussed in the light of literature reports.

KEY WORDS: Extrapyramidal system, multiple sclerosis, parkinsonism.

Introduction

On rare occasions, movement disorders have been reported in addition to the clinical findings commonly seen in multiple sclerosis (MS). Of these movement disorders, tremor is the one most frequently reported. Paroxysmal involuntary movements such as dystonia or tonic spasms have been well defined in MS, while an association with persistent choreathetosis, ballism and parkinsonism has been less frequently encountered (1,2). In MS, demyelinating plaques are usually distributed in the periventricular and/or subcortical white matter. Since the subcortical gray matter also contains myelinated nerve fibers, plaques can also be found in the striatum, pallidum and thalamus. This may be the pathological basis for the other movement disorders seen in a small proportion of patients with MS (1). This report discusses parkinsonism in a patient with definite MS (the relapsing-remitting type).

Case report

The history of a fifty-five-year-old female patient included an episode of sudden visual loss that responded well to corticosteroids (in 15 days) when she was 36 years old. At the age of 39, she was diagnosed with MS after developing weakness and numbness in the lower extremities, a diagnosis supported by positive magnetic resonance imaging (MRI) findings. She did not accept corticosteroid treatment, and her symptoms thus continued without her daily life being disrupted by intervening paroxysmal vertigo, nausea and vomiting. At the age of 45, she had a severe right lower extremity paresis that developed over a period of 5 days, and almost totally regressed after high dose intravenous steroid therapy. At the age of 48, numbness and stiffness in the legs and sphincter dysfunction appeared, later resolved with low dose corticosteroids. At that stage, she was seen by us and her neurological examination revealed right temporal pallor in the optic disk, left cerebellar signs and increased deep tendon reflexes in the right lower extremity. Cranial MRI showed periventricular, corpus callosal, right cerebellar and pontine multiple demyelinating lesions. Her clinical state was stable until the age of 51. Then, she noticed general slowing in movements, impairment of hand skills and, later, hand tremor. Her neurological examination showed bilateral temporal pallor in the optic disk, bilateral dysmetria, mild paresis of both arms, and brisk reflexes in all extremities. Besides these findings, she had static tremor affecting both hands, bradydysmia, bradykinesia, an anteflexed posture and decreased arm swing. She was admitted to hospital with these findings and given levodopa (3x62.5 mg/d), which resulted in a marked improvement. There was no worsening of other neurological findings and no change in her Expanded Disability Status Scale (EDSS) scores. At this point, steroid treatment was not considered and her cranial MRI showed an additional demyelinating lesion without gadolinium enhancement in the mesencephalon, including the substantia nigra (Fig. 1). At her 4th year follow-up, she was stable clinically with regard to the extrapyramidal and other neurological signs and symptoms.
Discussion

Paroxysmal movement disorders can accompany clinically established MS, but non-paroxysmal movement disorders are much more uncommon accompaniments to the disease (1,3). Postural and action tremors commonly occur in patients with MS and are thought to occur due to involvement of the cerebellar pathways. Cerebellar or brainstem involvement, or both, are seen in 81.6% cases of MS at some time during the illness (4). However, other types of movement disorder, such as tonic spasms and paroxysmal dystonia, are not commonly seen in MS. There have been reports of demyelinating lesions observed in the basal ganglia, thalamus and subthalamic nucleus as a result of which the above symptoms have been considered to be associated with MS (1,5). Others have reported that neuroradiologic findings do not show a strong anatomic correlation. Therefore this association has also been suggested to be coincidental (6-8).

The association of parkinsonism with MS has been reported in a total of eleven patients (1,3,4,9,10-12). Of these cases, two had only resting tremor, while nine had akinesia and rigidity. Five of these nine patients also displayed resting tremor. Four patients responded to levodopa well while three experienced drug-induced dyskinesia. One patient did not respond to levodopa; however, the parkinsonian symptoms improved with corticotherapy (10). This result was interpreted as an indication that there could be a pathophysiological association between MS and parkinsonism (9,10). Federlein et al. (10) noticed the simultaneous appearance of a lesion near the substantia nigra and correlating extrapyramidal motor symptoms in an MS patient with parkinsonism. After observing a clear improvement in the extrapyramidal symptoms following corti-
cotherapy in this patient, these authors suggested that parkinsonism in MS might result from transient functional disturbances in the substantia nigra or dopaminergic pathways of MS sufferers. On the other hand, in a pathological study, Kamphorst and Ravid reported that 3 out of 17 MS patients showed Lewy bodies in the brainstem, and nigral cell loss similar to that seen in Parkinson’s disease. Interestingly none of these patients had extrapyramidal symptoms (13). Even though these authors suggested that this finding may point to a causal or promoting relationship between MS and Parkinson’s disease, others suggested that the association between MS and parkinsonism is likely to be coincidental (1,9).

Our patient with bradykinesia, bradydymia, anteflexed posture and resting tremor also had cerebellar, pyramidal system and optic nerve involvement and a typical demyelinating disease history. There are several reasons for considering this case as a coincidence of idiopathic Parkinson’s disease and MS. First, her parkinsonism appeared much later than her MS symptomatology, second, she responded to levodopa therapy in a way similar to that seen in idiopathic Parkinson’s disease, and third, many MS patients with plaques in the midbrain, including the substantia nigra, do not manifest parkinsonian features. Therefore we suggest that our patient is a coincidental case of MS and idiopathic Parkinson’s disease. However, in order to understand whether the relationship between these symptoms is merely coincidental or causal, there is a need for histopathological studies in similar cases and investigations of parkinsonism in larger series of MS patients. This approach would make it possible to reach more definitive conclusions on this matter.

References