



A Rare Case of Right Hemiparkinsonism Due to Demyelination Disorder: Case Report

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ABSTRACT

Hemiparkinsonism, characterized by tremor, rigidity, and bradykinesia affecting one side of the body, can occur as a secondary, often subacute, symptom of demyelinating diseases like multiple sclerosis or osmotic demyelination syndrome. We reported a very rare case of right hemiparkinsonism due to demyelination disorder in a 47-year-old female from south India. A 47-year-old female presented with a history of hemiparkinsonism characterized by imbalance while walking. She was having normal blood pressure and blood sugar levels. She was doing well and able to do her activities of daily living. CSF for oligoclonal band revealed type 2 band pattern oligoclonal bands seen only in CSF, while no bands were seen in serum indicating intrathecal IgG synthesis. However, tests for anti-aquaporin-4 (NMO) IgG antibodies and Anti-MOG IgG antibodies showed negative. Her contrast MRI brain scan suggestive of multiple chronic cerebral and brainstem demyelinating plaques. Additionally, the left frontoparietal and right parietal periventricular lesions with paramagnetic rim signs indicate chronic active (smouldering) lesions. Furthermore, abnormal VEP showed bilateral optic nerve demyelination. The patient condition of right sided hemiparkinsonism secondary to demyelinating disorders improved on treatment with neuro anti-inflammatory drugs. In conclusion, to the best of our literature this is the very rare case of right sided hemiparkinsonism secondary to demyelination disorders presented with clinical manifestations of imbalance while walking in a 47-year-old female from south India.

Keywords: Hemiparkinsonism; Demyelination disorder; Multiple sclerosis; Walking imbalance; Plaques.

Introduction

Parkinson's disease remains the leading cause of parkinsonism and is a neurodegenerative condition marked by progressive loss of dopaminergic neurons within the pars compacta of the substantia nigra (SNpc) [1]. Nevertheless, parkinsonism is not exclusively linked to idiopathic dopaminergic neuronal degeneration and may arise from diverse underlying mechanisms [2]. A rare but recognized cause of hemiparkinsonism is mechanical compression of the basal ganglia. Various space-occupying intracranial

lesions—including tumors, large arachnoid cysts, giant aneurysms, and chronic subdural hematomas—have been reported to produce parkinsonian features [3-6].

Hemiparkinsonism is defined by unilateral manifestations such as tremor, rigidity, and bradykinesia, and may occur as a secondary, often subacute, presentation in demyelinating disorders like multiple sclerosis or osmotic demyelination syndrome. Although parkinsonism is typically associated with idiopathic Parkinson's disease, in demyelinating conditions it results from focal lesions involving the

basal ganglia or the nigrostriatal pathways [7]. Herein we reported a very rare case of right sided hemiparkinsonism secondary to demyelination disorder in a 47-year-old female.

Case Presentation

A 47-year-old woman from South India presented with features of hemiparkinsonism, characterized by imbalance while walking. Her vital and metabolic parameters—including blood pressure (110/70 mmHg), fasting blood glucose (79 mg/dL), and postprandial blood glucose (123 mg/dL)—were within normal limits. She was functionally independent in activities of daily living (ADL). Her clinical examination revealed that patient was consciousness and oriented with normal MMSE score (30/30), mild slurring of speech, right side rigidity, bradykinesia, and mild dragging of the right foot (Video 1) along with Unified Parkinson's Disease Rating Scale (UPDRS) score of 22. A demyelinating disorder was suspected. Her cerebrospinal fluid (CSF) analysis for oligoclonal bands demonstrated a type 2 pattern, with bands present exclusively in CSF and absent in serum, indicating intrathecal IgG synthesis. However, testing for anti-aquaporin-4 (NMO) IgG and anti-MOG IgG antibodies was negative.

Contrast-enhanced MRI of the brain revealed ill-defined FLAIR hyperintensities in the infratentorial region, involving the anterior and posterior margins of the pons, the periaqueductal region, and the peripheral anterolateral midbrain. Additional small FLAIR

hyperintense foci were noted in the left brachium pontis and left superior cerebellar peduncle. The remainder of the brainstem, cerebellum, and fourth ventricle appeared normal. The supratentorial imaging showed multiple T2/FLAIR hyperintense lesions in the bilateral fronto-temporo-parieto-occipital periventricular deep white matter and juxtacortical regions, without diffusion restriction. Several lesions exhibited T1 hypointense “black holes.”

Periventricular lesions were oriented perpendicular to the lateral ventricles, consistent with Dawson's fingers. A distinct right parietal periventricular lesion demonstrated a thin hypointense rim on susceptibility-weighted imaging (SWI), indicative of the paramagnetic rim sign. Similar findings were present in two lesions within the left frontal and parietal periventricular regions. A small, well-defined lesion was also identified in the posterior limb of the left internal capsule. The remaining cerebral parenchyma, ventricular system, midline structures, sella turcica, cranial nerves, and intracranial vasculature were unremarkable.

Overall, MRI findings were suggestive of multiple chronic demyelinating plaques involving both the cerebrum and brainstem (Figure 1). The presence of paramagnetic rim signs in select lesions indicated chronic active (smouldering) pathology. Additionally, visual evoked potential (VEP) testing revealed bilateral optic nerve demyelination (Figure 2).



Figure 1. Brain MRI: Suggestive of multiple chronic cerebral and brainstem demyelinating plaques.

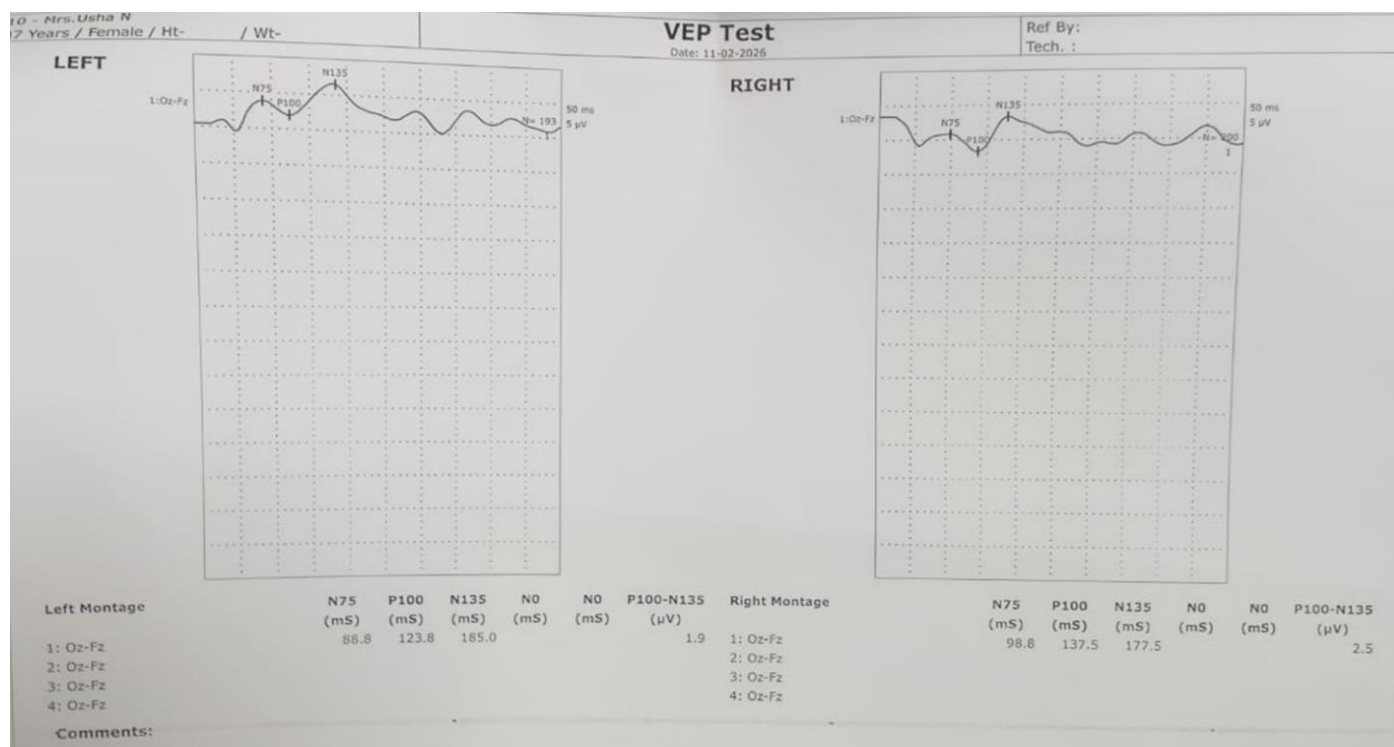


Figure 2: VEP test findings.

The patient condition of right sided hemiparkinsonism due to demyelination disorders improved on treatment with inj. methyl prednisolone 1 gm infusion for 5 days followed by oral prednisolone 50 mg per day along with Levo dopa and carbidopa and Trihexy phenidyl 4 mg per day. Patient was also started on Tab. Dimethyl fumarate 250 mg twice a day. The patient gradually made a significant improvement, able to walk better with reduction in rigidity and bradykinesia (Video 2), and her UPDRS score before treatment and post-treatment was 22 and 8 respectively.

Discussion

Parkinson's disease is an age-related disorder; defined by deep gray matter abnormality. Malfunction in the motor loop of the basal ganglia loop leads to Parkinsonism. Motor circuit includes substantia nigra, neostriatum, subthalamic nucleus, globus pallidus, and motor nuclei of thalamus (mostly ventral anterior and ventrolateral nuclei) [8]. Lesions of the basal ganglia could cause Parkinsonism manifestations [9]. Studies have shown that impairment of the blood-brain barrier, infiltration of the peripheral immune within this barrier and finally, inflammation in the CNS have important roles in Parkinson's disease pathogenesis [10], similar to what happens in multiple sclerosis. One of the supporting pieces of evidence implying on this claim is the reduction of Parkinson's disease developing risk in individuals who are taking anti-inflammation drugs [11].

Multiple sclerosis is an autoimmune demyelination disease that commonly causes white matter plaques, but deep grey matter nuclei can also be affected. Multiple sclerosis manifests with a wide range of neurologic symptoms depending on the location of lesions. Parkinsonism is rarely comorbid with multiple sclerosis. Parkinsonism associated with multiple sclerosis has been previously reported in the literature implicated as both coincidental and causal [12,13]. Coincidentally occurring parkinsonism with multiple sclerosis is supported by absence of demyelinating lesions in the basal ganglia on MRI, with abnormal uptake on DaTSCAN [13-15]. Evidence for a causal relationship includes a strategically placed plaque in the basal ganglia-thalamocortical network that is time locked to the onset of parkinsonism, which subsequently improves with high-dose corticosteroids [12,16,17].

While, in our case a right sided hemiparkinsonism secondary to demyelination disorder was observed showing multiple chronic cerebral and brainstem demyelinating plaques on contrast MRI brain scan. Furthermore, left frontoparietal and right parietal periventricular lesions with paramagnetic rim signs were suggestive of chronic active (smouldering) lesions. Literature evidences proved that due to the existence of myelinated fibers in gray matter, multiple sclerosis can affect cortical/subcortical gray matter; especially in chronic phases of the disease. Furthermore, low contrast between demyelination areas

and normal tissues because of low density of myelin in gray matter leads to the existence of invisible lesions, which cannot be relieved with usual MRI in most cases. More sensitive MRI methods such as double inversion recovery show gray matter lesions five times more than common conventional MRI method. However, almost 80% of gray matter lesions detected by microscopic examinations cannot be observed either by this technique. Studies demonstrated that gray matter lesions are common in all clinical courses of multiple sclerosis. Furthermore, in some cases, gray matter lesions are formed earlier than white matter ones [18].

In conclusion, to the best of our literature this is the very rare case of right sided hemiparkinsonism secondary to demyelination disorders presented with clinical manifestations of imbalance while walking in a 47-year-old female from south India.

Patient Consent Declaration

Authors hereby declare that they have obtained patient consent.

Conflict of Interest

None

Funding

None to declare.

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