CASE REPORT

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Levodopa-Responsive Parkinsonism After Bilateral Hemorrhagic Stroke

İki Yanlı Hemorajik İnme Sonrası Gelişen Levodopa Yanıtlı Parkinsonizm Olgusu

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ABSTRACT

Movement disorders after stroke are not uncommon and frequently manifest as chorea, ballismus, tremor, or parkinsonism. Post-stroke movement disorders may present as an acute symptom of the cerebrovascular event or, in certain instances, may emerge as a delayed sequela. This case report aimed to examine parkinsonism in a 55-year-old female patient who experienced two hemorrhagic strokes approximately five years apart and demonstrated a positive response to levodopa therapy. It is important to note that, in addition to pyramidal signs, extrapyramidal signs may also be observed subsequent to a cerebrovascular accident, and levodopa therapy can significantly ameliorate clinical manifestations.

Keywords: Hemorrhagic stroke, parkinsonism, movement disorders, levodopa, case report

ÖZ

İnme sonrası hareket bozuklukları nispeten yaygındır ve sıklıkla kore, ballismus, tremor, parkinonsizm şeklinde ortaya çıkmaktadır. İnme sonrası hareket bozuklukları kimi zaman inmenin akut semptomu olarak ortaya çıkarken bazı durumlarda ise gecikmiş bir sekel olarak ortaya çıkabilir. Bu olgu sunumunda, yaklaşık 5 yıl arayla iki kez hemorajik inme geçiren 55 yaşındaki bir kadın hastada gelişen ve levodopa tedavisine yanıt veren parkinsonizm ele alınmaktadır. İnme sonrası piramidal bulguların yanı sıra ekstrapiramidal bulguların da görülebileceği ve levodopa tedavisinin klinik bulguları anlamlı ölçüde düzeltebileceği akılda bulundurulmalıdır.

Anahtar Kelimeler: Hemorajik inme, parkinsonizm, hareket bozuklukları, levodopa, olgu sunumu

INTRODUCTION

Post-stroke movement disorders are one of the most common causes of secondary movement disorders[1]. The incidence is estimated to be approximately 1-4% among all strokes[2-4]. Post-stroke movement disorders may present as parkinsonism or a variety of hyperkinetic disorders such as chorea, ballism, athetosis, dystonia, tremor, myoclonus, stereotypy, and akathisia[5]. Such disorders can present as acute symptoms of the stroke or may emerge later as delayed sequelae[6,7]. Here, we aimed to present a case of post-stroke parkinsonism following a hemorrhagic stroke, which showed a favorable clinical response to levodopa therapy.

CASE REPORT

A 55-year-old female patient presented to our outpatient clinic with complaints of bradykinesia and speech disturbance following second episode of hemorrhagic stroke. Her medical history included hypertension and two episodes of hemorrhagic stroke. The patient experienced the first hemorrhagic stroke 5 years ago, and the second took place three months prior to presentation; both episodes required hospitalization. Neurological examination revealed dysarthric speech, hypomimia, bilateral wrist rigidity, and bilateral bradykinesia. Her gait was characterized by short steps with reduced

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associated arm swing bilaterally. There were no complaints of constipation, rapid-eye-movement disorders or hyposmia as the non-motor symptom associated with idiopathic Parkinson's disease.

Brain magnetic resonance imaging demonstrated a T2-weighted fluid attenuated inversion recovery hyperintensity in the right putamen consistent with a subacute hematoma (Figure 1A.), as well as bilateral diffuse T1 hypointensities in the putaminal white matter (Figure 1B). Following the initiation of levodopa/benserazide (25/100 mg, three times daily), a significant improvement in parkinsonian symptoms was observed. This clinical improvement was sustained at both the 6-month and 12-month follow-up visits.

DISCUSSION

Parkinsonism can develop as part of a degenerative process or, as in our case, due to secondary causes[7,8]. In secondary parkinsonism, core features such as bradykinesia, rigidity, resting tremor, and postural instability, commonly seen in primary Parkinson's disease, are also present[4,7,9]. Additionally, dysarthria, hypomimia, and gait disturbances, as observed in our patient, may accompany the clinical presentation. However, unlike idiopathic Parkinson's disease, the findings in our case were bilateral, and no non-motor symptoms were observed. In stroke-related cases, coexisting neurological deficits may further complicate the clinical picture[6]. In our patient, who exhibited clinical signs of stroke, newly manifested extrapyramidal signs were addressed, and treatment for parkinsonism was initiated.

In the literature, hyperkinetic disorders such as chorea, ballism, or dystonia, which are more pronounced and easily recognized, are frequently reported early in the post-stroke period[3,5]. In contrast, hypokinetic disorders like parkinsonism often have a more insidious onset or may be masked by other neurological impairments, potentially delaying diagnosis[1,10]. Therefore, clinicians should consider parkinsonism in stroke survivors presenting with acute or gradually worsening bradykinesia, rigidity, or gait disturbances.

Classical vascular parkinsonism typically results from chronic or recurrent ischemic lesions due to small vessel disease, usually with a gradual onset and predominant lower-body involvement[11]. Hemorrhagic strokes, however, can acutely or subacutely involve the basal ganglia or deep gray matter, leading to parkinsonism[6]. In our case, the subacute recognition of parkinsonism and the patient's presentation with notable functional impairment suggest a clinical course that differs from "classical" vascular parkinsonism. While patients with vascular parkinsonism often do not benefit significantly from levodopa, this patient demonstrated marked improvement in parkinsonian symptoms with levodopa therapy despite bilateral striatal damage. Indeed, similar cases of levodopa-responsive parkinsonism have been reported in isolated bilateral putaminal hemorrhage though they are relatively rare[12].

CONCLUSION

Most cases of post-stroke movement disorders reported in the literature have been associated with lesions involving small vessel disease in the middle cerebral artery or posterior cerebral artery territories[11,13]. However, cases of parkinsonism

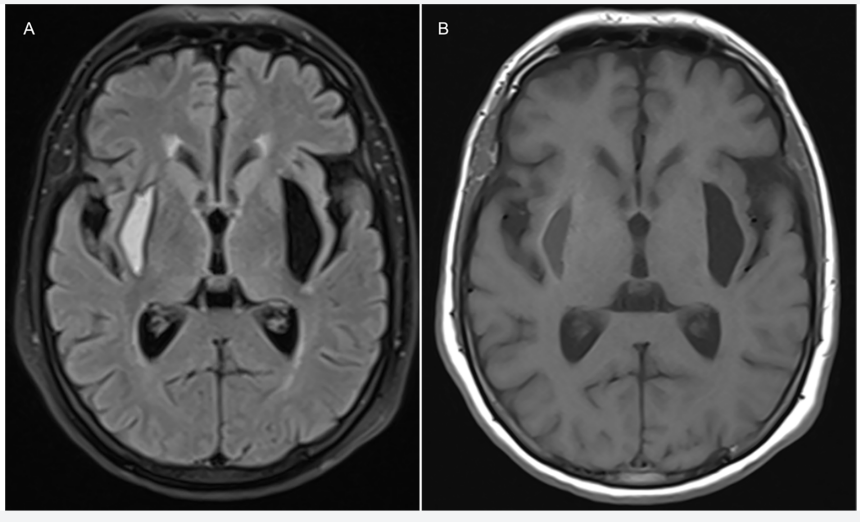

Figure 1. Brain magnetic resonance imaging demonstrated a T2-FLAIR hyperintensity in the right putamen consistent with a subacute hematoma (A), as well as bilateral diffuse T1 hypointensities in the putaminal white matter (B)

due to acute midbrain infarctions have also been reported[14]. These vascular territories encompass the basal ganglia structures, playing a critical role in the pathogenesis of post-stroke movement disorders. Furthermore, hemorrhagic strokes are emphasized in the literature to have a higher propensity to cause movement disorders compared with ischemic strokes likely due to more prominent tissue damage and inflammatory responses in the basal ganglia and extrapyramidal system[6]. In our patient, lesions in the bilateral putamen explained the acute-subacute course of parkinsonism.

In stroke survivors, it is crucial to recognize extrapyramidal involvement in addition to classic pyramidal signs. Hypokinetic movement disorders such as parkinsonism, which considerably impact quality of life, should not be overlooked. Treatment plans including both pharmacological interventions and rehabilitation strategies must be individualized to optimize patient outcomes.

Ethics

Informed Consent: Written informed consent was obtained from all participants.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Y.E., Concept: S.K., Y.E., Design: S.K., Y.E., Data Collection or Processing: S.K., Y.E., Analysis or Interpretation: S.K., Y.E., Literature Search: S.K., Y.E., Writing: S.K., Y.E.

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REFERENCES

1. Tater P, Pandey S. Post-stroke movement disorders: clinical spectrum, pathogenesis, and management. Neurol India. 2021;69:272-83.
2. Alarcón F, Zijlmans JC, Dueñas G, Cevallos N. Post-stroke movement disorders: report of 56 patients. J Neurol Neurosurg Psychiatry. 2004;75:1568-74.
3. Ghika-Schmid F, Ghika J, Regli F, Bogousslavsky J. Hyperkinetic movement disorders during and after acute stroke: the Lausanne Stroke Registry. J Neurol Sci. 1997;146:109-16.
4. Handley A, Medcalf P, Hellier K, Dutta D. Movement disorders after stroke. Age Ageing. 2009;38:260-6.
5. Mehanna R, Jankovic J. Movement disorders in cerebrovascular disease. Lancet Neurol. 2013;12:597-608.
6. 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.
7. Kim JS. Delayed onset mixed involuntary movements after thalamic stroke: clinical, radiological and pathophysiological findings. Brain. 2001;124:299-309.
8. Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry. 2008;79:368-76.
9. Adams RD, Victor M, Ropper AH. Adams and Victor's Principles of Neurology. McGraw-Hill Education. 2019;11.
10. Freedman M, Sarkar G, Hill MD. Movement disorders following cerebrovascular events. Can J Neurol Sci. 2010;37:234-8.
11. Bansil S, Prakash N, Kaye J, Wrigley S, Manata C, Stevens-Haas C, et al. Movement disorders after stroke in adults: a review. Tremor Other Hyperkinet Mov (N Y). 2012;2:tre-02-42-195-1.
12. Hatano T, Kubo S, Niijima-Ishii Y, Hattori N, Sugita Y. Levodopa-responsive parkinsonism following bilateral putaminal hemorrhages. Parkinsonism Relat Disord. 2013;19:477-9.
13. Bogousslavsky, J. Post-stroke movement disorders: a clinical and imaging study. European Neurology. 2003;49:133-9.
14. Friedman-Korn T, Weill C, Ben-Haim S, Arkadir D. Delayed levodopa-responsive parkinsonism following acute midbrain injury. J Neurol Sci. 2024;459:122983.