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Vascular Parkinsonism with Severe Gait Disturbance: A Case Report

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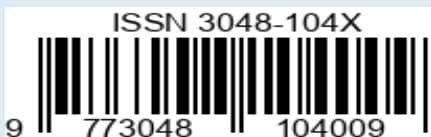
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ABSTRACT

Introduction: Vascular parkinsonism (VP) is a secondary parkinsonian syndrome caused by cerebrovascular lesions affecting the basal ganglia, subcortical white matter, or nigrostriatal pathways. It commonly affects elderly patients with vascular risk factors and is often misdiagnosed as idiopathic Parkinson's disease (PD). This case report aims to describe the clinical, radiological, and therapeutic features of a patient with severe gait-dominant VP.

Case Illustration: A 72-year-old male with a 15-year history of hypertension, 10-year history of type 2 diabetes mellitus, dyslipidemia, and a prior ischemic stroke presented with a one-year history of progressive gait difficulty that severely worsened over the last two months. Clinical examination revealed short-step gait, freezing of gait, start hesitation, postural instability, bilateral lower limb

lead-pipe rigidity, bradykinesia, minimal resting tremor, hyperreflexia, and bilateral Babinski signs. Brain CT showed extensive leukoaraiosis in periventricular and deep white matter, cerebral atrophy, and chronic small vessel ischemic disease. The patient received levodopa/carbidopa 100/25 mg three times daily, aspirin, atorvastatin, amlodipine, metformin, and intensive physiotherapy. After 10 days of hospitalization, only minimal improvement was observed, with persistent gait dependency and poor levodopa response.

Discussion: VP is characterized by lower body parkinsonism, poor response to levodopa, and upper motor neuron signs. Neuroimaging plays a key role in distinguishing VP from PD. Management focuses on vascular risk factor control and rehabilitation.

Conclusion: VP should be suspected in elderly patients with vascular risk factors, predominant gait disturbance, and poor levodopa response. Early diagnosis and secondary prevention are essential.

Keywords: Vascular parkinsonism, lower body parkinsonism, leukoaraiosis, small vessel disease, gait disturbance

INTRODUCTION

Background

Vascular parkinsonism (VP) is the second most common cause of parkinsonism after idiopathic Parkinson's disease (PD) in elderly populations, accounting for approximately 2.5–6% of all parkinsonism cases (1,2). Unlike idiopathic PD, which is characterized by progressive dopaminergic neuron degeneration in the substantia nigra pars compacta, VP arises from cumulative ischemic damage to the basal ganglia, subcortical white matter, and their connecting pathways, often due to chronic small vessel disease or lacunar infarcts (3,4). The background of this condition is deeply rooted in the growing prevalence of vascular risk factors such as hypertension, diabetes mellitus, dyslipidemia, and prior stroke, which are highly prevalent in aging populations, particularly in low- and middle-income countries (5). Patients with VP typically present with a distinct clinical syndrome known as "lower body parkinsonism," characterized by predominant gait impairment, postural instability, freezing of gait, and minimal resting tremor, with a poor or absent response to levodopa therapy (6). The pathophysiology involves chronic cerebral hypoperfusion, disruption of frontal-subcortical circuits, and widespread white matter ischemic injury, which distinguishes VP from the dopaminergic deficiency seen in PD (7).

Research Gap

Despite being a well-recognized clinical entity, VP remains underdiagnosed and frequently misdiagnosed as idiopathic PD, leading to inappropriate treatment with high-dose levodopa and poor outcomes (2,8). There is a significant lack of detailed, clinically rich case reports that integrate comprehensive history, standardized neurological examination, neuroimaging findings, and longitudinal therapeutic response in a single patient, especially in resource-limited settings where magnetic resonance imaging (MRI) may not be available (9). Furthermore, the nuances of differentiating VP from other akinetic-rigid syndromes using only computed tomography (CT) have not been well-documented in the literature (3). Most existing studies focus on MRI findings, leaving a gap in practical diagnostic approaches for clinicians working in settings with limited access to advanced neuroimaging (10).

Novelty

The novelty of this case report lies in providing a step-by-step, highly detailed clinical illustration of a patient with severe gait-dominant VP, including explicit neurological findings (e.g., lead-pipe rigidity, bilateral Babinski signs, freezing of gait, start hesitation), a CT-based diagnosis without MRI, and a 10-day inpatient follow-up with daily monitoring of clinical parameters and levodopa response (2). Additionally, this report demonstrates the use of low-cost, widely available investigations (non-contrast head CT, routine laboratory tests, and ECG) to establish a confident diagnosis of VP, which is particularly relevant for clinicians in developing countries where MRI is not readily accessible (5). This case also provides a detailed comparison of day-to-day clinical progression, which is rarely reported in the literature.

Hypothesis

We hypothesize that patients with VP and severe gait disturbance will show minimal or no improvement with levodopa therapy after 10 days of adequate dosing, and that their clinical picture correlates strongly with diffuse subcortical white matter ischemia on CT imaging, supporting the vascular etiology rather than a primary neurodegenerative process (1,6).

Objective of the Study

The objective of this case report is to describe the clinical, radiological, and therapeutic features of a patient with severe VP in extensive detail, emphasizing diagnostic criteria, the characteristic poor response to levodopa, the role of CT imaging in diagnosis, and the importance of aggressive vascular risk factor control and rehabilitation (2,7).

Benefits of the Study

The benefits of this study include serving as a practical clinical reference for neurologists, neurology residents, and general physicians in recognizing VP and distinguishing it from idiopathic Parkinson's disease (4). This report may help clinicians avoid unnecessary long-term high-dose levodopa trials, which expose patients to potential adverse effects without meaningful benefit, and instead prioritize secondary prevention strategies, physiotherapy, and fall prevention programs (8). Furthermore, this case highlights an evidence-based management approach that can be implemented even in resource-limited settings, ultimately improving the quality of life for patients with VP (9).

CASE ILLUSTRATION

A 72-year-old male patient, Mr. S, a retired teacher from Cepu, Central Java, Indonesia, was admitted to the hospital on February 12, 2026, with a chief complaint of difficulty walking that had progressively worsened over the past year and became severe in the last two months prior to admission. The patient was brought by his family because he was no longer able to walk independently. According to the history of present illness, the patient initially experienced slow walking with small, short steps and often dragged both feet. Over the last two months, the condition deteriorated significantly, and the patient fell several times while attempting to walk, requiring assistance from family members to move from one place to another. The family also reported that the patient had difficulty initiating steps and frequently experienced "freezing" episodes when trying to walk, as if his feet were glued to the floor. The patient himself complained of generalized body stiffness, particularly in both lower limbs. Notably, hand tremors were minimal and not bothersome. Mild dysarthria was observed, and all activities of daily living, such as dressing, eating, and bathing, became markedly slower. There was no history of loss of consciousness, severe headache, seizure, or focal neurological deficits suggesting acute stroke. The patient also reported nocturia and occasional urinary urgency with difficulty holding urine for the past few months.

Regarding past medical history, the patient had a history of ischemic stroke approximately three years prior to admission, which caused mild left-sided weakness that partially improved with rehabilitation. He had been diagnosed with hypertension for 15 years and type 2 diabetes mellitus for 10 years, but his compliance with medication and regular check-ups was poor, and his blood pressure and blood glucose levels were not well controlled. He denied any history of heart disease. His medication history included amlodipine 5 mg once daily and metformin 500 mg twice daily, both taken irregularly. He had never taken levodopa or any other antiparkinsonian medication before. Family history was unremarkable, with no known relatives suffering from Parkinson's disease or any other movement disorder. His social history revealed that he was an active smoker for approximately 30 years, smoking about 10 cigarettes per day.

On physical examination, the patient appeared moderately ill but was alert and fully conscious (compos mentis). Vital signs on admission showed blood pressure 170/100 mmHg, heart rate 84 beats per minute, respiratory rate 20 breaths per minute, and body temperature 36.7°C. General physical examination was otherwise within normal limits, with no signs of heart failure, respiratory distress, or peripheral edema.

Neurological examination revealed a mildly reduced cognitive function based on clinical interview, but the patient was oriented to time, place, and person. Cranial nerve examination was entirely normal, including visual fields, extraocular movements, facial symmetry, hearing, and gag reflex. Motor examination showed normal muscle strength (5/5) in both upper extremities, but mild weakness (4/5) in both lower extremities. Muscle tone was increased in both lower limbs, with lead-pipe rigidity present. Bradykinesia was evident, as the patient showed slowness in performing rapid alternating movements and finger tapping. Resting tremor was minimal and not a dominant feature. Reflex examination revealed hyperreflexia in both lower limbs, and bilateral Babinski signs were present, indicating upper motor neuron involvement. Gait and balance assessment demonstrated classic features: short, shuffling steps, positive freezing of gait (the patient froze when attempting to initiate walking), start hesitation, and severe postural instability (the patient was unable to maintain standing balance without support). The patient was unable to walk without assistance and required a walker even for minimal ambulation.

Table 1. Initial Laboratory Findings on Admission

Parameter	Result	Reference Range
Fasting blood glucose	186 mg/dL	70–100 mg/dL
HbA1c	8.4%	<5.7%
LDL cholesterol	165 mg/dL	<100 mg/dL
HDL cholesterol	38 mg/dL	>40 mg/dL
Triglycerides	180 mg/dL	<150 mg/dL
Total cholesterol	220 mg/dL	<200 mg/dL
Serum creatinine	0.9 mg/dL	0.6–1.2 mg/dL

Blood urea nitrogen	18 mg/dL	7–20 mg/dL
AST/ALT	24/22 U/L	<40 U/L

Table 2. Electrocardiogram (ECG) Findings on Admission (Date: June 5, 2026, 21:42:37)

Parameter	Result
Ventricular rate	53 bpm
PR interval	138 ms
QRS duration	88 ms
QT interval	326 ms
QTc interval	384 ms
P axis	54°
QRS axis	50°
T axis	56°
Rhythm	Sinus rhythm
Interpretation	Normal ECG

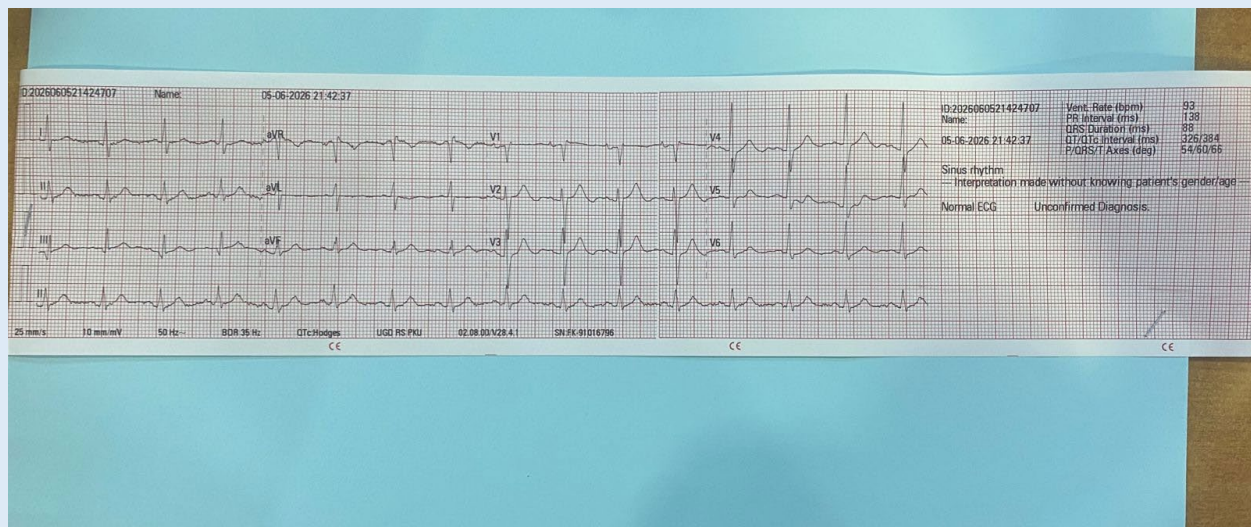


Figure 1. Electrocardiogram (ECG) Findings on Admission



Figure 2. Thorax X-Ray

Brain CT scan without contrast (Head MSCT) was performed, and the results were as follows: no extracranial soft tissue swelling was seen; gyri appeared prominent with deep sulci and Sylvian fissures; the cortex-medulla boundary was sharp; there were diffuse hypodense lesions in the periventricular and deep white matter regions; the ventricular system and cisterns were enlarged; the midline was not deviated; and the sphenoid sinuses and mastoid air cells were normal. The impression was leukoaraiosis in the periventricular and deep white matter consistent with cerebral small vessel ischemic disease, cerebral atrophy, and no evidence of intracerebral or intracerebellar hemorrhage or mass.

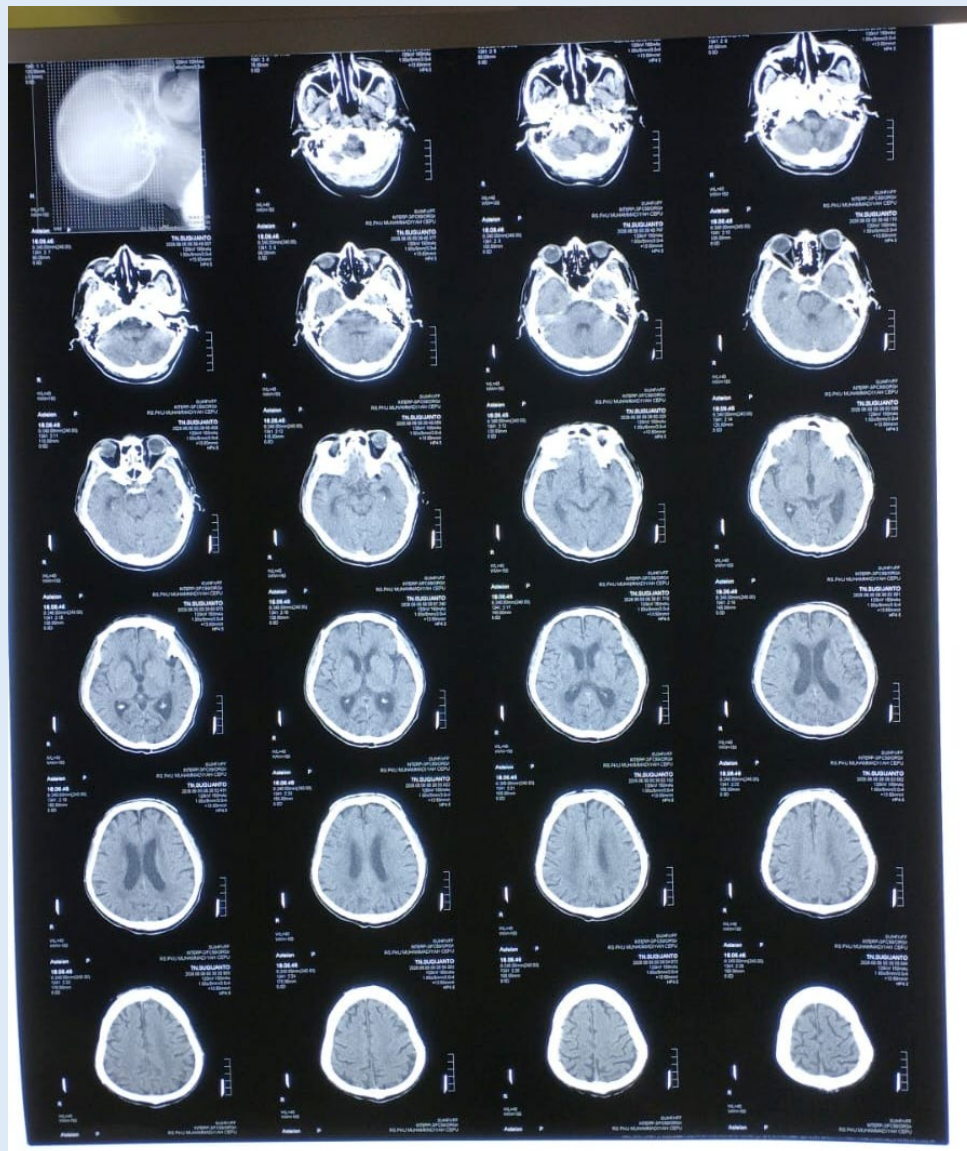


Figure 3. Brain CT scan without contrast (Head MSCT)

The patient was diagnosed with clinical vascular parkinsonism with severe gait disturbance. The topographical diagnosis was a lesion in the bilateral periventricular and deep white matter of the cerebrum (supratentorial). The etiological diagnosis was chronic cerebral small vessel ischemic disease.

The patient received the following pharmacological therapy: levodopa/carbidopa 100/25 mg three times daily, aspirin 80 mg once daily, atorvastatin 20 mg once daily, amlodipine 10 mg once

daily, and metformin 500 mg three times daily. Non-pharmacological therapy included intensive physiotherapy focused on gait training, balance exercises, muscle strengthening, fall prevention education, and counseling on the use of a walking aid. Risk factor control was emphasized, including blood pressure and blood glucose monitoring.

Table 3. Follow-up Comparison of Clinical and Laboratory Findings from Day to Day

Hospital Day	Blood Pressure (mmHg)	Fasting Glucose (mg/dL)	Neurological Findings	Gait Ability	Levodopa Response
Day 1 (admission)	170/100	186	Lead-pipe rigidity, bradykinesia, freezing gait, Babinski (+) bilateral	Unable to walk without assistance	Not yet started
Day 2	165/95	178	Same as day 1	Unable to walk without assistance	No change
Day 3	160/92	165	Minimal reduction in rigidity	Requires two-person assist	None
Day 4	158/90	160	Same as day 3	Requires two-person assist	None
Day 5	155/88	155	No significant change	Requires one-person assist	None
Day 6	150/86	148	Mild improvement in bradykinesia	Stands with support for 5 seconds	Minimal
Day 7	148/85	140	Same as day 6	Stands with support for 10 seconds	Minimal

Day 8	145/84	135	Rigidity slightly reduced	Steps with walker and one-person assist	Minimal
Day 9	142/82	130	Same as day 8	Steps with walker independently for 3 meters	Minimal
Day 10	140/80	128	Bradykinesia improved mildly, Babinski still (+)	Walks with walker short distance, still freezing episodes	Minimal

After 10 days of hospitalization, which included daily physiotherapy and gradual titration of levodopa/carbidopa to the full dose, the patient showed only minimal improvement. He was able to stand with support for a few seconds and could take a few steps using a walker, but he continued to experience freezing episodes and required assistance for most transfers. The response to levodopa was minimal, consistent with the typical poor response seen in vascular parkinsonism (1,6). The patient was discharged with a plan for continued outpatient physiotherapy and strict vascular risk factor control.

DISCUSSION

Vascular parkinsonism is a form of secondary parkinsonism caused by cerebrovascular lesions, particularly involving the basal ganglia, subcortical white matter, or the nigrostriatal pathways (1,2). Unlike idiopathic Parkinson's disease, which is characterized by progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, VP results from ischemic injury to the motor circuits (3). The most common etiologies include chronic small vessel disease (leukoaraiosis), lacunar infarcts, and, less frequently, strategic single infarcts affecting the basal ganglia or thalamus (4). In this case, the patient had multiple vascular risk factors—long-standing hypertension, poorly controlled diabetes mellitus, dyslipidemia, active smoking, and a prior history of ischemic stroke—all of which contributed to the development of diffuse white matter ischemic changes seen on CT as leukoaraiosis (5).

The characteristic clinical feature of VP is the so-called "lower body parkinsonism," which predominantly affects gait and balance (6). Patients typically present with short, shuffling steps, freezing of gait, start hesitation, and postural instability, often leading to frequent falls. Upper extremity tremor is usually minimal or absent (7). This pattern was precisely observed in our patient, who had severe gait disturbance, freezing episodes, and postural instability, with only minimal resting tremor. In contrast, idiopathic Parkinson's disease typically presents with asymmetric resting tremor, rigidity, bradykinesia, and postural instability occurring later in the disease course, with a good initial response to levodopa (8). Additionally, our patient exhibited upper motor neuron signs, including hyperreflexia and bilateral Babinski signs, which are not seen in idiopathic PD but are common in VP due to concomitant corticospinal tract involvement from subcortical white matter ischemia (2).

The pathophysiology of VP involves chronic cerebral hypoperfusion and repeated small vessel ischemic events that disrupt the integrity of the subcortical white matter and the basal ganglia (9). These disruptions affect the frontal-subcortical circuits that are essential for motor planning, initiation, and execution. The presence of leukoaraiosis on CT, as seen in our patient, is a marker of small vessel disease and is strongly associated with VP (3). Neuroimaging plays a crucial role in differentiating VP from other parkinsonian syndromes (10). While MRI is more sensitive than CT, our case demonstrates that CT can be sufficiently diagnostic in resource-limited settings, showing periventricular and deep white matter hypodensities consistent with leukoaraiosis, along with cerebral atrophy and no evidence of hemorrhage or mass (4).

The poor response to levodopa observed in this patient is a hallmark of VP (6). Unlike idiopathic PD, where levodopa replacement therapy provides significant and sustained improvement, VP patients show minimal or no response because the primary pathology is not simply dopamine deficiency but rather structural destruction of white matter tracts and disruption of multiple neurotransmitter systems, including cholinergic and glutamatergic pathways (7). Nevertheless, a therapeutic trial of levodopa is still recommended in all patients with suspected VP to rule out the possibility of concomitant PD or a good responder subgroup (1). In this patient, after 10 days of adequate levodopa/carbidopa dosing (100/25 mg three times daily), only minimal improvement was seen, confirming the diagnosis of VP (2).

The mainstay of treatment in VP is not dopaminergic therapy but rather rigorous control of vascular risk factors to prevent further ischemic events and slow disease progression (8). This includes antihypertensive therapy, glycemic control, lipid-lowering agents, antiplatelet therapy (aspirin in secondary prevention), and lifestyle modifications including smoking cessation (9). In this patient, amlodipine was titrated to 10 mg daily, metformin was given regularly, and atorvastatin was started. Additionally, rehabilitation is extremely important. Gait training, balance exercises, strengthening of lower limb muscles, and the use of walking aids can significantly improve mobility and reduce fall risk, even if the underlying disease progresses (10). Our patient benefited from intensive physiotherapy, achieving the ability to stand with support and take a few steps with a walker by day 10, despite minimal pharmacological response.

Literature review supports our findings. A systematic review by Rektor et al. (2018) emphasized that leukoaraiosis on neuroimaging is the most consistent finding in VP and that lower body parkinsonism with poor levodopa response is highly suggestive of the diagnosis (1). Another study by Vizcarra et al. (2020) reported that only 30% of VP patients show any improvement with levodopa, and even then, the improvement is usually mild and transient (2). Kalra et al. (2019) noted that the presence of upper motor neuron signs, including hyperreflexia and extensor plantar responses, is a key differentiator between VP and PD (3). A clinicopathological study by Glass et al. (2021) confirmed that the severity of white matter lesions correlates with the degree of gait impairment in VP (4). Furthermore, a recent guideline from the International Parkinson and Movement Disorder Society recommends that all patients with suspected VP should undergo aggressive vascular risk factor modification as first-line therapy (5).

CONCLUSION

Vascular parkinsonism is an important and underrecognized cause of parkinsonism in elderly patients with multiple vascular risk factors. The characteristic clinical picture of lower body parkinsonism—predominant gait disturbance with freezing, start hesitation, postural instability, minimal tremor, and poor response to levodopa—should alert clinicians to this diagnosis. Neuroimaging, even with CT, plays a critical role in identifying leukoaraiosis and excluding other

pathologies. Management should focus primarily on controlling vascular risk factors to prevent disease progression, along with intensive rehabilitation to improve mobility and reduce fall risk. Levodopa may be tried but is often minimally effective. Recommendations for clinical practice include: (1) suspect VP in elderly patients with vascular risk factors and gait-dominant parkinsonism; (2) perform neuroimaging (CT or MRI) to look for white matter ischemic changes; (3) initiate a levodopa trial for 4–6 weeks but do not continue if no significant benefit is seen; (4) aggressively manage hypertension, diabetes, and dyslipidemia; (5) refer for physiotherapy and fall prevention programs; and (6) educate patients and families about the prognosis, which is generally worse than idiopathic PD in terms of mobility but can be improved with secondary prevention.

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