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LETTER TO EDITOR

Postural Instability Due to Right Hemisphere Stroke and the Role of Cognitive Functions

Dear Editor,

A 55-year-old female patient with a history of hypertension and diabetes mellitus presented with a severe gait impairment that had developed following an ischemic stroke of the inferior division of the right middle cerebral artery 2 months previously. She exhibited no motor paralysis or cerebellar disturbances, so she was referred to the neurology department for the investigation of other possible etiologies of the gait problem.

On neurological examination, the patient was found to be cooperative and fully orientated. Remarkably, the visual field examinations showed visual field loss on the left side of the vertical midline. The findings of the motor and cerebellar investigations were within normal limits. However, the sensory investigations revealed hypoesthesia over the whole left side of the body, and the sensory and visual neglect subtypes were also present. In addition, the patient's deep tendon reflexes were normoactive, while her pathological reflexes (Babinski sign, Hoffman sign, etc.,) were absent, which indicated that the pyramidal tract was not disturbed. However, the patient could not mobilize without support due to experiencing a severe imbalance. Her postural stability was severely impaired (she scored four points on the Retropulsion test, as defined by the Movement Disorder Society-Unified Parkinson's Disease (PD) Rating Scale). Due to the severe postural instability, she had fallen several times over the past 2 months, which had left her with a significant fear of falling [Videos 1-2]. Based on these findings, the patient's gait pattern was evaluated as being a dyspraxic gait accompanied by severe postural instability, which had occurred due to a recent stroke affecting the right temporoparietal lobe. The findings of the cranial magnetic resonance imaging (MRI) showed a diffusion restriction in the right temporal lobe, superior parietal lobule, and angular gyrus [Figure 1]. However, a chronic ischemic lesion in the right occipital lobe was also identified [Figure 2]. The findings of the etiological investigations concerning the early-onset stroke, including computed tomography brain/neck angiography, echocardiography, and rhythm Holter, were all within normal limits. Yet, tests concerning the patient's vasculitis biomarkers revealed positive results for antinuclear antibody anticardiolipin antibody immunoglobulin G (IgG), and beta-2 glycoprotein IgG.

Following interconsultation with the rheumatology department, a diagnosis of antiphospholipid syndrome (as the etiological agent behind ischemic stroke) was established and warfarin treatment was initiated. Besides, based on published data highlighting the importance of supraspinal control and certain cognitive functions during locomotion, further cognitive tests were performed to determine the specific cognitive profile of the postural instability and gait dyspraxia observed in our patient. She scored 20 points on the standardized mini-mental state examination. The patient could only count four numbers forward and count backward from two-digit numbers, which indicated a severe impairment in her attention and executive functions. In addition, she scored 12 points on the Benton Face Recognition Test (two standard deviations below the mean), which indicated an impairment in relation to object discrimination that is likely associated with the injury to her right posterior hemisphere, especially the parietal and occipito-parietal lobes. Furthermore, she could not perform any task included in the judgment of the line orientation test. Remarkably, she scored zero points on the watch drawing test, and she could not even copy elementary figures [Figure 3]. Furthermore, the single letter cancellation test revealed both a left-sided visual deficit and visual spatial neglect [Figure 4].

DISCUSSION

In our patient, the findings of the motor and cerebellar examinations were all within normal limits. Although the presence of left-sided hemihypoesthesia was determined, we classified the gait pattern and postural instability of the patient as a higher-level gait disorder, which could be associated with disturbances in the supraspinal control center of locomotion. Higher-level gait disorders (or gait dyspraxia) represent rather less common subtypes, and this particular type of gait disorder being triggered by a stroke is even rarer in the literature.

In our previous reports, two cases with gait dyspraxia in whom the causative lesion sites included the right occipital lobe were illustrated.[1,2] However, in this newly reported patient, postural stability was the most prominently disturbed function, and it was the main component of both the dyspraxia and the falling problems. Therefore, we prefer to define the patient as having "postural instability due to stroke." A disruption in postural control can be involved in clinical manifestations during the acute and chronic phases of stroke.[3,4] The study by Peurala *et al*. found that patients with left hemiparesis and right

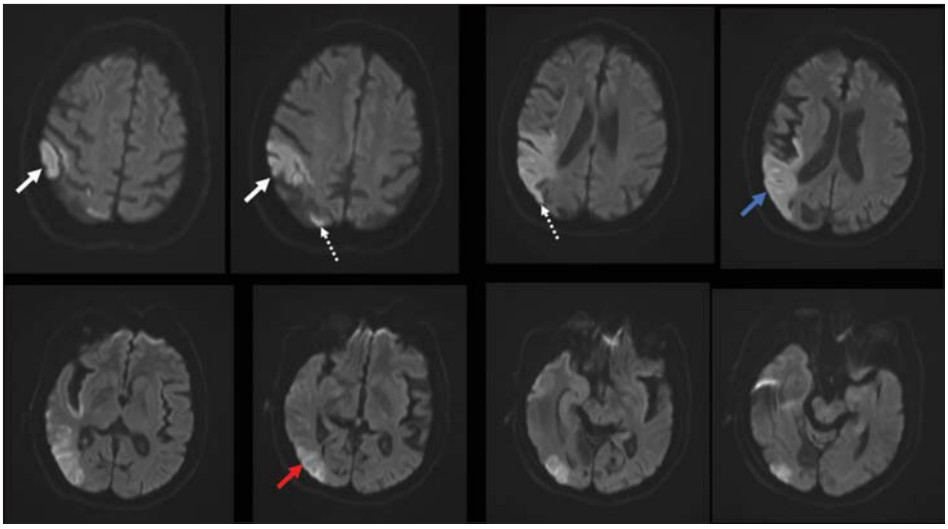

Figure 1: Diffusion-weighted imaging sequences showing high signal in the right superior (white arrows), middle (blue arrow), and inferior temporal gyrus (red arrow) and superior parietal lobule (ragged arrows)

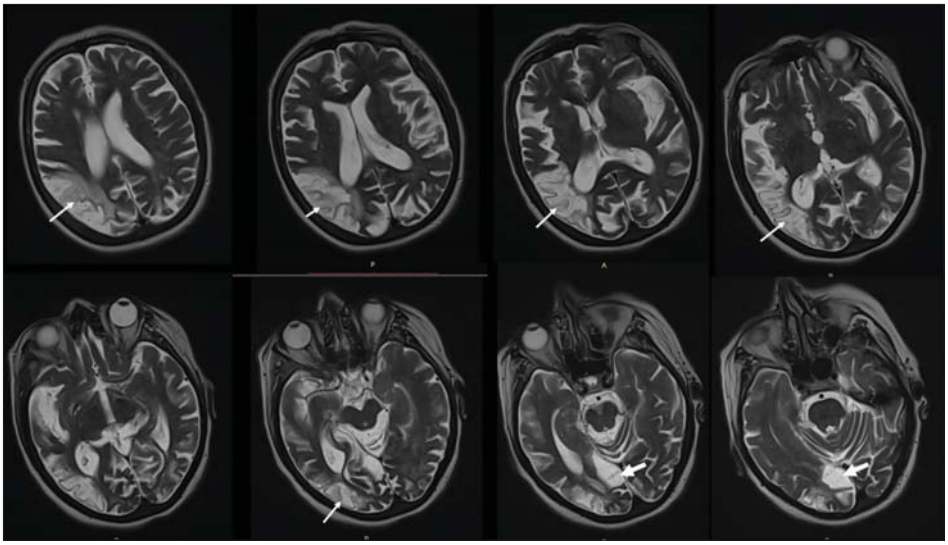

Figure 2: T2-weighted magnetic resonance imaging sequences performed 2 months after stroke, showing ischemic lesion in the right temporoparietal lobe (arrows) and sequela lesion in the right occipital lobe (thick arrows)

hemisphere stroke experienced postural disturbances more prominently.[3] However, despite this finding, the responsible anatomical regions and the cognitive correlates of postural instability in stroke patients have not yet been investigated in detail.

On the one hand, postural instability is recognized as a cardinal motor symptom of PD, which usually emerges during the later stages of the disease. On the other hand, it can also be present in other neurological diseases, including Parkinson-plus syndromes, normal pressure hydrocephalus, vascular parkinsonism, and Alzheimer's disease.[5] Postural instability appears as a tendency to be unstable when standing, and a person experiencing postural instability may easily fall backward if slightly jostled. It is considered to be one of the most disabling symptoms of PD due to its causal association with an increased risk of falling.[6] A substantial body of literature has recognized the relationship between global cognitive dysfunction and the motor symptoms of postural instability/gait disturbance in PD.[7-9] In addition, PD patients with the postural instability/gait disturbance subtypes have been shown to be at a higher risk of developing dementia.[9] In the crucial report by Uc *et al*., poorer visuospatial abilities and diminished executive functioning were both found to be associated with worse gait and postural instability in PD

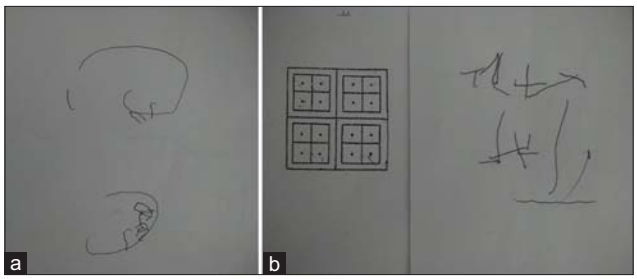

Figure 3: The imaged showing the performance of the patient in the clock drawing test (a) and figure copying test (b)

patients without dementia.[10] In the recent report by Kelly *et al*., the impairment of global cognition and different aspects of executive functioning was found to be associated with more severe postural instability symptoms. For this reason, the authors hypothesized the existence of multiple neural pathways that contribute to the associations between cognition and postural instability/gait disturbance symptoms in patients with PD.[11] Wang *et al*. confirmed the results of Kelly *et al*. in their large-group of patients with PD.[11,12]

However, to the best of our knowledge, no large-scale study has yet addressed the cognitive profile of postural instability in patients with vascular dyspraxia. In our previous reports, two cases with vascular dyspraxia accompanied by right occipital infarcts were illustrated.[1,2] However, postural instability was not the most prominent finding and widespread cognitive tests (as in this case) were not performed. Taken together, we believe that this case represents a crucial illustration in this regard. In accordance with the results of reports concerning PD patients, a widespread cognitive impairment (most prominently in the visuospatial functions as well as in the attention and executive functions) was determined in our index cases.

A major discussion may be the possible role of a primary sensory deficit of left-sided visual defect and neglect in the disturbed locomotion pattern of the patient. However, there was no asymmetrical impairment in her gait and the prominent disturbance was the postural instability which suggest a disruption of more complex cognitive networks related with the right hemisphere functions rather than the injury of primary sensory modalities.

In conclusion, through the illustration of this rare case, we discuss the potential role of widespread cognitive control in the execution of postural stability and locomotion. Future reports of larger case series including patients with postural instability of vascular origin may provide crucial contributions regarding our understanding of the physiology of locomotion.

Consent for publication

Informed consent form has been obtained.

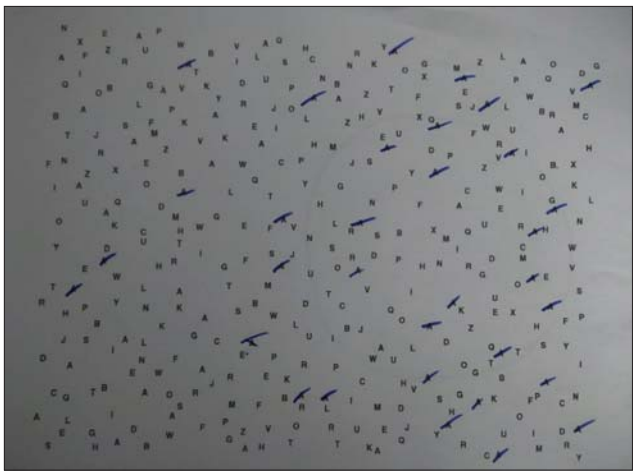

Figure 4: The single letter cancellation test result of the patient showing the left-sided visual deficit and visual spatial neglect

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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Videos available on: www.jmedscindmc.com

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