A neglected symptom? Parietal gait lateropulsion as primary manifestation of acute is-chemic stroke

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Introduction. Gait disorders are commonly overlooked as a presenting manifestation of stroke and underrepresented in case series. We describe four cases of sudden-onset gait lateropulsion as primary manifestation of parietal lobe stroke.

Case report. Four patients presented after sudden-onset gait lateropulsion. On neurological examination, all patients had at least one cortical sensory deficit and wide-based gait with lateropulsion towards the side of the cortical deficit. Neuroimaging revealed a subacute parietal lobe stroke contralateral to the side of gait lateropulsion. In two patients we found bilateral lateropulsion with predominance towards the side of cortical deficit and increase of unsteadiness with eye closure (an apparent Romberg sign), with neuroimaging revealing bilateral parietal strokes (subacute contralateral and chronic ipsilateral to gait lateropulsion).

Conclusion. We report gait lateropulsion as a novel primary manifestation of acute stroke of the parietal lobe (parietal gait lateropulsion). Given its role as the destination of proprioceptive pathways, parietal strokes can result in gait lateropulsion, with bilateral lesions even mimicking sensory ataxia with bilateral lateropulsion and unsteadiness upon eye closure.

Key words. Gait ataxia. Gait lateropulsion. Ischemic stroke. Neurologic gait disorders. Parietal ataxia. Parietal lobe.

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Introduction

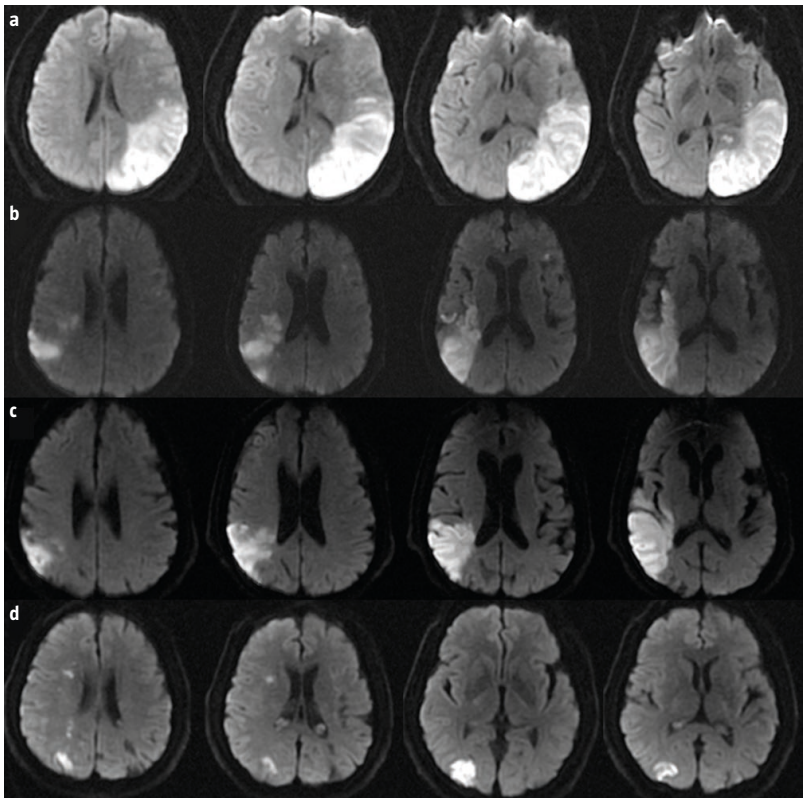
Gait lateropulsion results from unilateral cerebellar or vestibular disease, or from bilateral cerebellar or dorsal column disease. However, since the parietal lobe represents the destination of cerebellar and dorsal column pathways, a localized parietal stroke could mimic an injury to these structures, but isolated parietal strokes represent less than 1% of cases in large series. Clinical manifestations include sensory loss (either primary or cortical), visuospatial neglect, and, less frequently, ataxia [1], but gait impairment remains underreported as initial manifestation of stroke. We report four cases of parietal lobe stroke with gait lateropulsion as presenting manifestation.

Cases report

Case 1. 42-year-old female with long-standing arterial hypertension presents to the emergency department 12 hours after sudden-onset right sided lateropulsion of gait along and right hemibody numbness. On neurological examination, her speech is non-fluent with preserved comprehension, semantic paraphasic errors, and abolished nomination and repetition, alexia, and agraphia. Cranial nerve examination is unremarkable. Motor exam reveals right upper limb dysmetria, dysdiadochokinesis, upward-drift, Stewart-Holmes phenomenon, with normal strength and hyperactive deep-tendon-reflexes. Primary sensory modalities (thermoalgesic and proprioception) were preserved, but cortical loss could not be evaluated due to language impairment. A wide-based gait, with right lateropulsion and absent Romberg sign. Magnetic resonance imaging revealed an extensive stroke of the left parieto-occipital region, reaching far to the external capsule and retrocapsular region (Fig. 1a).

Case 2. 79-year-old male with long-standing diabetes *mellitus* and arterial hypertension presents to the emergency department 72 hours after sudden-onset left sided lateropulsion of gait, along with confusion and discrete left hemiparesis. On neurological examination, he is found with temporospatial disorientation with preserved language and left visuospatial hemineglect. Cranial nerve examination reveals a discrete left hemifacial supranuclear palsy. On motor examination, we found a

Figure 1. Magnetic resonance imaging findings. Case 1 (row a): extensive stroke of the left parieto-occipital region, reaching far to the external capsule and retrocapsular region. Case 2 (row b): subacute stroke involving the right angular gyrus, and adjacent centrum semiovale. Case 3 (row c): subacute ischemic stroke in the right parieto-occipital junction, with the involvement of the angular region of the parietal lobe, with ischemia reaching the semioval center. Case 4 (row d): subacute stroke involving the right postcentral and angular gyri.



left ataxic hemiparesis with hyperreflexia, and the sensory exam revealed left hemibody a decreased thermoalgesic perception without cortical sensory deficit. Gait was wide-based with left-lateropulsion and absent Romberg sign. Magnetic resonance imaging revealed a subacute stroke involving the right angular gyrus, and adjacent centrum semiovale (Fig. 1b).

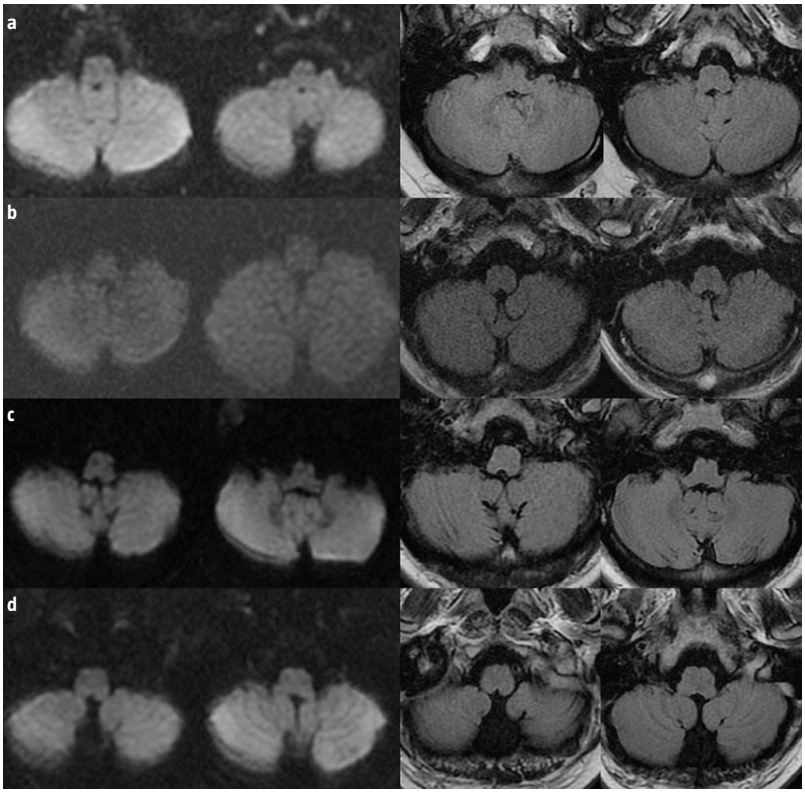
Case 3. 63-year-old male chronic smoker with long-standing diabetes *mellitus* and arterial hypertension presents to the emergency department 24 hours after sudden-onset left sided lateropulsion of gait, along lack of awareness of symptoms (as reported by the family). On neurological examination, he is found alert with intact higher mental functions. Cranial nerve examination revealed only left homonymous hemianopia. Left hemibody dysmetria and dysdiadochokinesis, with normal strength and deep-tendon-reflexes. Sensory examination found normal thermoalgesic and proprioceptive sensation with impaired graphesthesia, stereognosis and multimodal (sensory and visual) extinction. Gait was wide-based with predominantly left-lateropulsion and inability to perform tandem gait, with increased unsteadiness after eye closure (Romberg sign). Magnetic resonance imaging revealed a subacute ischemic stroke in the right parieto-occipital junction, with the involvement of the angular region of the parietal lobe, with ischemia reaching the semioval center; also, a contralateral chronic stroke of the parieto-occipital junction is found (Fig. 1c).

Case 4. 59-year-old male with long-standing arterial hypertension and a left-hemispheric stroke (secondary to carotid artery stenosis that underwent endarterectomy) three years ago presents to the emergency department 24 hours after wake-up-onset of indistinct lateropulsion of gait and left hemibody hypesthesia and hemiparesis. On neurological examination, he is found alert with intact higher mental functions. Cranial nerve examination reveals left hemifacial hypesthesia to all sensory modalities and left facial supranuclear palsy. Left hemiparesis (Medical Research Council scale, 4/5), with dysmetria, dysdiadochokinesis (disproportionate to weakness), upper limb upward-drift and Stewart-Holmes phenomenon. Left-sided decreased sensitivity to light touch, sparing thermalgesic sensation without a cortical sensory deficit. Gait was wide-based gait, with indistinct lateropulsion, inability to perform tandem gait, and present Romberg sign. Magnetic resonance imaging revealed a subacute stroke involving the right postcentral and angular gyri, with a contralateral chronic stroke involving the precentral and postcentral gyri (Fig. 1d).

The patients were consecutive patients admitted to the neurovascular care unit because of acute ischemic stroke, between April and December of 2018. In all cases, imaging demonstrated a parietal subacute ischemic stroke (Fig. 1) contralateral to the side of gait lateropulsion, without cerebellar or frontal lobe involvement (Fig. 2); in cases with indistinct lateropulsion and apparent Romberg sign, a contralateral parietal chronic stroke was found (Fig. 3). All the patients had a pre-stroke modified Rankin scale of 0 and none were under the influence of sedative or hypnotic medications prior to stroke onset. Etiological assessment of stroke revealed carotid artery stenosis (case 4), with the re-

mainder (cases 1, 2, and 3) classified as potential embolic strokes of unknown source [2], after work-up ruled out large-artery disease (intra and extracranial atherosclerosis or dissection) through carotid ultrasound and computed tomography angiography of the cervical and cerebral vessels, cardiac embolism through normal 24-hour Holter monitoring and transthoracic echocardiogram, and small-vessel disease (previous lacunar strokes or microbleeds) was not present in the magnetic resonance imaging. Cases 2 and 4 showed recovery of gait and sensory abnormalities during hospitalization and were discharged with a modified Rankin scale 2 after completion of etiological assessment; whereas cases 1 and 3 had progression of stroke extension despite statin and antiplatelet agent therapy and were discharged with a modified Rankin scale 4.

Figure 2. Absence of cerebellar involvement. Difussion weighted imaging and T_2 FLAIR sequences demonstrating absence of subacute or chronic lesions in the cerebellum of all patients.



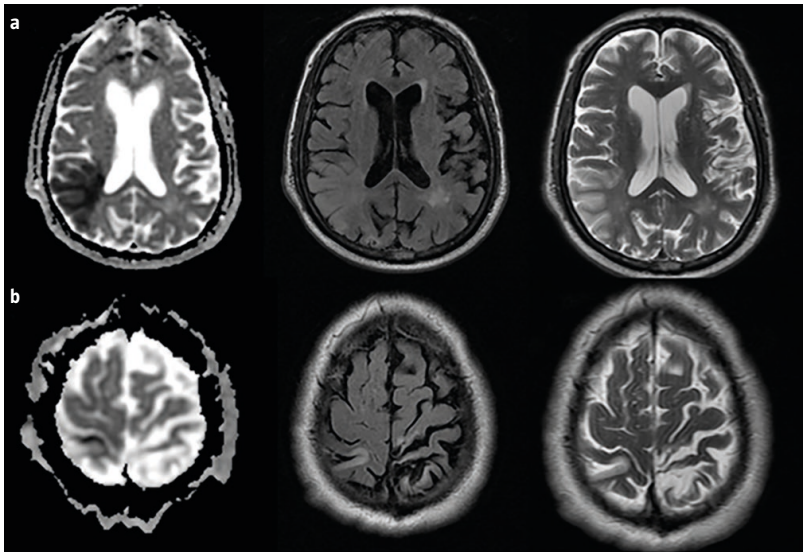
Discussion

To our knowledge, gait lateropulsion had not been reported as a primary manifestation of parietal ischemic stroke. Existent case-series of stroke restricted to the parietal lobe have focused on reporting either motor or sensory manifestations [3,4]. However, these studies did not evaluate gait, probably because initial evaluations of stroke patients are based on the National Institutes of Health Stroke Scale which does not include a formal examination of gait. It was recently demonstrated that lateropulsion is not uncommon after stroke [5,6]. When evaluated 30 days after hemispheric stroke, contralesional lateropulsion was present in 48% of patients after right-sided stroke, whereas it affected only 13% of patients after left-sided stroke [6]. A further in-depth analysis of the components of lateropulsion suggested that this manifestation might represent a form of spatial neglect [5].

Ataxia due to parietal injury was first reported by Gerebetzoff in1938, and classified as 'pseudo-cerebellar' incoordination, since it was not associated with sensory loss [3]. Ghika et al described the clinical spectrum of the motor parietal syndrome, noting that ataxia could occur despite normal proprioception [4], and was associated with sensory loss in 75% of cases, with pure 'pseudo-cerebellar' ataxia in only 4% [7]. Futamura and Kawamura further detailed the spectrum of parietal ataxia, reporting that of 13 patients with 'pure¡ parietal ataxia, six had sensory ataxia [8], and suggested that parietal ataxia was distinguished by upper-limb predominance with slowness in finger-nose-finger test and classified it into 'cerebellar-like? (prominent dysdiadochokinesis), and 'sensory-like' (prominent worsening after eye closure) [8]. It is noteworthy that gait was not evaluated in any of these series.

Pathophysiological mechanisms behind parietal ataxia and gait lateropulsion remain elusive. Although initially explained as the result of parietal injury with proprioceptive loss, but this was refuted by reports of patients with ataxia and preserved proprioception [4], suggesting that it might be an acute manifestation of impaired sensory (tactile and proprioceptive) integration, in the manner of extinction or astereognosis. Later studies which correlated neurovascular anatomy to clinical manifestations reported that posterior parietal injuries resulted in pseudo-cerebellar ataxia, whereas anterior parietal injuries resulted in sensory ataxia [8]. This partly corroborated Futamura's suggestion that sensory ataxia resulted from damage to Brodmann's area 3a in the anterior parietal area (desti-

Figure 3. Contralateral parietal chronic stroke in patients with Romberg sign. Apparent diffusion coefficient maps and T_2 FLAIR sequences revealing a left parietal subcortical (case 3, row a) and cortical (case 4, row b) chronic stroke.



nation of dorsal column fibers); whereas pseudo-cerebellar ataxia resulted from damage to Brodmann's area 5 in the posterior parietal area (destination of cerebellum fibers) [8].

However, we consider that this explanation is only partially correct. Brodmann's area 3a does receive proprioceptive information from muscle spindles, thus its injury could imitate sensory ataxia [9]. Whereas Brodmann's area 5 plays a more complex role, involved in object-directed movement aided by proprioception [9], thus its injury could mimic sensory ataxia, as suggested by Ghika et al [7]. The suggestion by Futamura of Brodmann's area 5 receiving cerebellar fibers was based upon animal models, and a parieto-cerebellar pathway was only recently demonstrated in humans, in which the pathway was associated with cognition [10]. This could explain the scarcity of pure 'pseudo-cerebellar' ataxia in patients with parietal stroke.

We suggest the term parietal gait lateropulsion, as supported by the notion that parietal lobes are involved in gait because they represent the final destination of cerebellar and dorsal column pathways, and the fact that cases 3 and 4 had bilateral lateropulsion with worsening upon eye closure as a result of non-simultaneous bilateral parietal infarcts. And suggest classifying ataxia due to parietal injury upon phenomenological grounds as pseudo-sensory when proprioceptive loss and worsening upon visual input suppression predominate, and as pseudo-cerebellar when appendicular dysmetria and dysdiadochokinesis predominate.

Sharing Critchley's vision, we consider that each patient with parietal injury represents 'a minor essay in clinical research', which we must study thoroughly to contribute 'to our sum-total of knowledge' [1]. May these cases serve as a reminder for us not to neglect the parietal lobes when localizing the lesion in a patient with sudden onset of gait lateropulsion.

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¿Un síntoma negado? Lateropulsión parietal de la marcha como manifestación inicial de ictus isquémico agudo

Introducción. Los trastornos de la marcha no suelen considerarse dentro de las manifestaciones de presentación del ictus y están subrepresentados en las series de casos. Presentamos cuatro casos de lateropulsión de la marcha de inicio súbito como manifestación primaria de ictus del lóbulo parietal.

Caso clínico. Cuatro pacientes se presentaron tras el inicio súbito de lateropulsión de la marcha. En el examen neurológico, todos tenían al menos un déficit sensitivo cortical, marcha de base amplia con lateropulsión ipsilateral al déficit cortical. En la neuroimagen se corroboró un ictus subagudo parietal contralateral al lado de lateropulsión de la marcha. Dos pacientes tenían lateropulsión bilateral con predominio ipsilateral al déficit cortical e incremento de inestabilidad con los ojos cerrados (simulando signo de Romberg), en quienes la neuroimagen demostró un ictus parietal bilateral (subagudo contralateral, crónico ipsilateral al lado de lateropulsión de la marcha).

Discusión. Describimos la lateropulsión de la marcha como una nueva manifestación inicial de ictus agudo del lóbulo parietal (lateropulsión parietal de la marcha), contralateral al lado de desviación de la marcha. Dado el papel del parietal como destino de las vías de propiocepción, los ictus pueden originar alteraciones de la marcha, con lesiones bilaterales que semejan ataxia sensitiva con inestabilidad al eliminar la aferencia visual.

Palabras clave. Ataxia de la marcha. Ataxia parietal. Ictus isquémico. Lateropulsión de la marcha. Lóbulo parietal. Trastornos neurológicos de la marcha.