International
Journal of Neurorehabilitation

Mini Review **Open Access**

Clinical and Pathophysiological Considerations of Gait Limitations and High Prevalence of Falls, in Elderly with Most Common, Disabling Neurological Diseases

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Abbreviations: PD: Parkinson's Disease; TD: Tremor Dominant; PIGD: Postural Instability Gait Disorder; FoG: Freezing of Gait; NIHSS: National Institutes of Health Stroke Scale; MS: Multiple Sclerosis; EDSS: Expanded Disability Status Scale; DTI: Diffusion-Tensor Tractography; WM: White Matter

Introduction

Falls in seniors represent an important cause of injury, disability, even death, and a major public health problem. Falling can indicate the decline of biological resources and of functional ability, resulting from cumulated multiple specific disabilities and chronic health problems [1].

Gait and balance disturbances are age-related and represent one of the most common gerontoneurological symptoms, with different etiology and clinical features: antalgic, paretic, spastic, ataxic, sensory deficit, hypokinetic, dyskinetic, anxious, and psychogenic. The pathogeneses of gait disorders include sensory deficits (visual, vestibular, somatosensory), neurodegenerative processes (cortical, extrapyramidal, cerebellar), toxic factors (medications, alcohol), depression and anxiety (primary or concerning falls) [2].

Everyday walking in complex environments is correlated with simultaneous cognitive and executive performances, scanning and planning. Elderly performing a concurrent cognitive task when walking showed greater decrements of spatio-temporal gait parameters; for this reason, dual-task testing was integrated in the comprehensive geriatric assessment to predict falls in community-dwelling older adults [3].

Dysfunction of the complex interplay between eye movements and locomotion may contribute substantially to the occurrence of falls. Suppression of the vestibulo-ocular reflex is responsible for postural inadequacy and prone to falls in gerontoneurological patients with progressive supranuclear palsy or in Parkinson's disease (PD) [4]. Vestibular neglect indicates an inappropriate dissociation between poor vestibular perceptions during caloric stimulation (despite a normal horizontal canal function) and an aberrant central processing of peripheral inputs, with lack of reactive postural corrections, and falls [5].

Physiopathological basis of gait and balance dysfunctions is better understood with modern neuroanatomical mapping technique. Cerebral white matter (WM) architectonics undergoes various degenerative changes with normal aging: decrease in myelin density and alterations in its structure [6].

Different topographic brain regions express different rates of change with aging, anterior brain being disproportionately more altered than posterior regions [7]. Fiber density mapping showed a significant correlation with age-related WM changes in the frontal lobes (prefrontal and precentral gyrus), limbic lobes (cingulate and parahippocampal gyrus), corpus callosum (genu) and the posterior limb of the internal capsule [8].

Selective and disproportionately anterior-to-posterior regional degradation of association fibers and commissural WM bundles (including cognitive networks) represent the biological substrate of age-related cognitive abasement, functional compromise in problem solving, declining of working memory, gait impairments, motor dyspraxia and increased risk for the development of neurodegenerative diseases [9,10].

Diffusion-tensor tractography (DTI) revealed a discriminative gender pattern of age's effect on microstructural topographic degradation of the human corpus callosum: in men, compromised fibers were mainly in genu; in women age degenerative effect was present in genu and splenium [11].

Owing to the neurological acute pathology or sequelae affecting integrative motor functions – like balance regulation and gait – falls frequency is particularly increased in neurogeriatric pathology, attributed to the pathological sensory-motor deficits and cognitive ones. Neurological disorders (stroke, neurodegenerative diseases), dementia, delirium, or psychotropic medication worsen the prognostic value of risk factors for future falls [12].

Stroke, dementia, disorders of gait and balance had an increased risk of falls (level A). Peripheral neuropathy, PD, lower extremity weakness or sensory loss, and substantial vision loss, probable had a risk of falls (level B). Falling in the past year strongly predicted the likelihood of future falls (level A) [13].

In a prospective survey including 228 independent community dwelling elderly with mild to moderate neurological impairments (treatable in outpatient facilities), incidence of falls was three times higher than in control participants, and particularly high post-stroke (89%), in PD (77%), dementia (60%) or epilepsy (57%). Neuropathies, peripheral nerve lesions and PD were predisposing conditions to recurrent accidental falling [14].

Another prospective study investigated 548 patients admitted to a neurological hospital (due to recurrence/ aggravation of symptoms), indicated 34% incidence of patients who felt once or more often during the last twelve months, double compared to age-matched population living in community. In chronically severe neurological patients, disturbance of gait was blamed for falling in 55% of seniors. Most frequent falls were registered in PD (62 %), syncope (57%) polyneuropathy (48 %), epilepsy (41 %), and spinal disorders (41 %). Paroxysmal attacks, loss of consciousness were responsible for 22% cases of falling (12% epileptic seizures, 10% syncope) [15].

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Falls are one of the most common medical complications post-stroke, occurring in all the evolutive phases (acute, rehabilitative, and chronic) [16]. Sequelary impairments represent stroke-specific risk factors for falls and influence short-time and long-time outcome; deficits of lower extremity strength, impaired balance, visual field deficits (visuospatial hemineglect), and self-care limitation, are better predictors of accidental falls than more general items such as age, incontinence, and sensory impairments [17]. Risk of falling in stroke survivors is more than twice compared to age-/or gender-matched controls. During the first 6 months after the cerebrovascular event, incidental falls were found in 32% [18] -73% subjects [19]. Consequences of post-stroke falls are: activity and participation limitation as a strategy to prevent recurrent events, increased dependence on caregivers, and the development of fear of falling. Experiencing a fall at the time of the stroke may have a direct impact on the development of fear of falling [18], inactivity and deconditioning, further increasing the danger of iterative falls (in a vicious circle) with negative repercussions on the quality of life.

Neurological deterioration of the strategic structures responsible for the supraspinal control of human gait, depression and cognitive problems represent pejorative factors for accidental falls. It is obvious that neurological impairment influence the propensity to fall; stroke survivors at risk (with moderate-severe disability) are rated with a NIHSS score ≥ 4 [20].

Hemiparetic gait is characterized by asymmetry, extensor synergy pattern of hip extension and adduction, knee extension, and ankle plantar flexion and inversion. It is a kaleidoscopic interplay of motor, sensory, proprioceptive loss, deficits of integrated representations of visual, somatosensory, and auditory peripersonal space (body neglect and environment/spatial neglect), spasticity and/or ataxia.

Gheorghe Marinescu, founder of the Romanian School of Neurology, realised in 1898 the first motion study of hemiparetic gait (Walking troubles of organic hemiplegia), followed by other masterpieces of "study with the help of the cinematograph" (Walking troubles of organic paraplegias – 1899, Progressive locomotor ataxia – 1900, Illnesses of the muscles – 1901, A case of hysteric hemiplegia healed through hypnosis – 1899) [21].

Corpulent stroke survivors have more alterations of kinematic walking parameters and are prone to falls [22]. Despite significant efforts focused on post-stroke gait retraining, during conventional rehabilitation, 33% of community-dwelling individuals continue to demonstrate walking asymmetries [23].

Patients with Parkinson`s disease (PD) fall more frequently than other old persons, due to motor complications, postural instability and gait disorders. Typical dysfunctional parkinsonian gait (described in 1817) is hypokinetic, with reduced stride length and velocity, shortening of the swing phase, increase of the stance phase, and longer double-limb support, start hesitation, shuffling and festinating gait, propulsion, and freezing of gait (FoG). Muscle tone rigidity and postural instability, faulty adjustment of postural muscle tone before gait initiation and absence of the rhythmic limb movements during walking, reduce walking capabilities in adapting to various environments, and predispose this category of patients to recurrent and severe falls. In addition to the disturbance of automatic movement processes, volitional and emotional expressions of movement processes are also seriously affected [24]. Attention may influence gait control, so any sensory (visual and/or auditory) cueing may temporary improve the stride length.

Many studies emphasized the clinically heterogeneity of PD [25-28].

The main subtypes, clustered mainly by clinical motor symptoms, are: the tremor dominant (TD), postural instability gait disorder (PIGD), respectively the indeterminate one. Overlapping between groups was seen in many objective features of gait and balance. Several independent observers have reported (in early and untreated PD subjects) specific "clusters of non-motor symptoms dominant phenotype of PD" [26]. Patients with the PIGD subtype (predominantly characterized by axial motor involvement and increased cognitive impairment) are predisposed to falls, contrasting with the TD subjects [27]. These clinical particularities raised the supposition of different neuropathology, genetic and etiologic influences, distinct neurotransmitter systems, and functional imaging aspects [25-28].

In a cohort of 207 newly diagnosed and drug naive PD patients, about 2% were classified as fallers and 15% as rare-fallers [29]. Accidental falls become a markedly increasing problem as the PD progresses. Falls occurred three times more frequently in PD (in 54% of PD vs. 18% of controls) [30]. Recurrent fallers (35%) had a significantly more severe motor disability [31], worse cognitive [27] and non-motor symptoms (greater general physical fatigue, less energy levels, depressive mood) than those who did not fall [32].

Cortical structures including the frontal and parietal lobes, the mesencephalic locomotor region and specifically, the pedunculopontine nuclear area, all apparently play important roles in the control of postural muscle tone and locomotion in PD [24,27]. FoG, remarkable in advanced phases of PD, might be associated with dysfunction of the fronto-basal ganglia circuit [24,33].

Patients with multiple sclerosis (MS) can have severe balance impairments (delayed responses to postural perturbations, increased sway in quiet stance, and reduced ability to regain the limits of stability), with severe repercussions on ambulation (decreased stride length and cadence, reduced gait speed). One-year prospective follow-up community-dwelling people with MS classified as fallers 61% of them [34].

Fall tendency may occur early in evolution, even before deterioration of locomotion and balance becomes obvious on clinical examination. Falling is correlated with a higher degree of disability, concretized by a significantly higher EDSS score (2.4 ± 1.4 versus 1.1 ± 0.9 in non-fallers) [35]. Evolutive loss of mobility and falls are the greatest prognostic concerns. The high prevalence of walking limitations noticed in this demyelinating disease have a tremendous impact on the functional status, basic and instrumental activities of daily living, and on the quality of life [36].

As mentioned afore, cognitive–motor interference generate practical problems in everyday life, including difficulties while walking and talking, potentially increasing the risk of falls. Individuals with MS performing a concurrent cognitive (dual-task) when walking, showed greater decrements of the spatio-temporal gait parameters (speed and swing time variability), compared to healthy controls. This executive dysfunctionality could be a result of divided attention deficit or overloading of the working memory system [37]; a poorer verbal memory can predict an increased risk for multiple falls [38].

Fear of falling and spatiotemporal gait alterations in people with MS are linked. Instrumented posturography demonstrated an elevated variability of the center of pressure trajectory, greater overall sway velocity and area, indicating disability deterioration and poor outcome [39,40]. Fearful patients walked slower, had a shorter step length, a wider base of support and prolonged double support phase compared to slightly concerned patients [41,42].

Quantitative fiber tracking studies (DTI) illustrated WM destructuration in parallel with the functional degradation, and permitted longitudinal evaluation of the myeloarchitectonics in MS [43].

Absolute T1 relaxometry emphasized topographic chronic tissue abnormalities of WM, with special focus on the microstructural organization of the corpus callosum, as an indicator for neocortical atrophy [44].

In elderly with cerebral small vessel disease, voxel-based morphometry detects different imagistic aspects: discrete lacunar infarcts and/or more diffuse regions of WM hyperintensities or leucoaraiosis. Conventional MRI and DTI emphasized the widespread disruption of WM integrity, responsible for the gait disturbances and cognitive impairment in lacunar stroke [45]. Extensive disconnections of bilateral cortical regions involved in executive control on motor performance, especially the prefrontal cortex (fibers in centrum semiovale and periventricular frontal lobe) were responsible for a lower gait velocity, shorter stride length and broader stride width [45]. Smoking aggravated the microstructural integrity of the cerebral WM (already affected by the small-vessel disease), and was associated with poorer cognitive functioning [46].

Conclusion

Elderly neurological outpatients are potentially at high risk for falls. A careful assessment of the multifactorial predisposing factors in both patient and environment, represents the first step of primary prevention measures. Clinically identifiable risk factors are: orthostatic hypotension, visual problems, impairment of gait or balance, medication, limitations in basic or instrumental activities of daily living, and cognitive degradation [1]. An adequate prophilaxis with multilevel corrective interventions at endogenous and /or exogenous levels, enrolling community-dwelling elderly in targeted prevention programs focused to raise awareness, represent the ”real cure” for injurious falling. For gerontoneurological impaired people, falling prevention is better than cure, and can considerably reduce the psychological and physical morbidity, and the dependence on family care, social support and healthcare services.

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