

## **Combined gait disorder: a diagnostic challenge –a case report**

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### **ABSTRACT**

Gait disorders are a major cause of functional impairment and morbidity, especially in the elderly population. Prevalence of gait disorders is higher in persons over 60: is estimated to be around 15% at 60 years of age and more than 50% in people > 80 years. Most gait disorders are multifactorial and have both neurologic and non-neurologic components. Neurological gait abnormalities result from focal or diffuse lesions occurring in the neural pathways linking the cortical motor centers to the peripheral neuromuscular systems. Nonneurological gait abnormalities include gait limitations caused by musculoskeletal, cardiac, or respiratory diseases. Assessment of a gait abnormality should include history, clinical presentation and additional diagnostic tests. Finding the ethiology of a gait disorder could be a challenge for the practitioners in many cases, requiring interdisciplinary cooperation.

**Key words:** gait disorder, neurologic evaluation, hyperuricemia

Walking relies on a complex, simultaneous interaction of the motor system, sensory control, and cognitive functions. Gait disorders are a major cause of functional impairment and morbidity, especially in the elderly population, where gait impairment is a common complain. Gait troubles increases the risk of falls, fractures, loss of autonomy, institutionalization and even death. Also, gait disturbances in the elderly are a risk factor for future cardiovascular disease and dementia.

Prevalence of gait disorders is higher in persons over 60: is estimated to be around 15% at 60 years of age and more than 50% in people > 80 years old [1]

Classification of gait abnormalities could be based on clinical features of gait (associated signs and symptoms) or on etiology of gait disorder (underlying cause) [2]

Risk factors for gait and balance disturbances include old age, vascular risk factors (e.g. high blood pressure or diabetes).

Occurrence of cerebrovascular disease in the elderly, particularly of cerebral small vessel disease, also appears to play some role in the development of gait and balance impairment.

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Non neurological gait abnormalities include gait limitations caused by musculoskeletal, cardiac, or respiratory diseases (eg arthritis, cardiac disease, chronic lung disease, and peripheral vascular disease).

In The Einstein Aging study which includes persons aged 70 and older, 35% of study participants were diagnosed with abnormal gaits: 41,6% presented with neurological, 48,2% with non-neurological and 10,1% with combined gait abnormalities [3]. In this population the prevalence of non-neurological gait abnormalities was higher

than the prevalence of neurological gait abnormalities, including combined (20.8% versus 15.7%). Causes of pure non-neurological gait abnormalities included in more than 80% of cases arthritis or joint deformities, and less frequent chronic lung disease, angina pectoris , cardiac failure and peripheral vascular disease. Causes of neurological gait troubles include ataxic gait - the most frequent, followed by hemiparetic , frontal, parkinsonian and neuropathic gait. [3].

In the population-based Bruneck Study cohort (2), which includes persons of 60 to 97 years, gait abnormalities have almost the same prevalence (32.2% of study population), but neurological gait disorders were more common (24.0% versus 17.4% non-neurological gait abnormalities) in this study population. In this study, non-neurological gait disorders were more frequent in females and occurred predominantly due to orthopaedic reasons.

Common causes of disturbed gait in the elderly are neurological deficits, including sensory deficits (e.g. peripheral neuropathy and vestibulopathy), neurodegeneration (e.g. cerebellar ataxia and parkinsonian syndromes), cognitive impairment (e.g. degenerative dementia), joint degeneration (e.g. coxarthrosis) and general loss of muscle mass (sarcopenia). Furthermore, a fear of falling also contributes to the gait disorder [4].

Assessment of a gait abnormality should include history, clinical presentation and additional diagnostic tests [5].

History should mention duration and course of gait disorder (episodic or continuous, sudden onset or slow progression), precipitating and exacerbating factors (darkness, fobia), accompanying symptoms and signs (dizziness, pain, etc), associated diseases (diabetes, heart failure), medication use and frequency of falls [6].

Clinical presentation allows recognition of a typical gait pattern, such as hemiparetic / spastic, ataxic, steppage, parkinsonian, dyskinetic, frontal, anxious or psychogenic gait [5]. This includes precise

clinical tests, such as “timed up-and-go test”, pull-test, measure of step length, walking speed, etc.

Additional paraclinical investigations most frequently used are neuroimaging studies, measure of nerve conduction velocities, vestibular and visual testing. New diagnostic modalities have evolved: dual tasking (patient is asked to walk while performing either a cognitive or a motor task), video gait analysis and functional neuroimaging (fMRI and <sup>18</sup>FDG-PET).

After using this complex algorithm in the management of a gait disorder, finding the precise cause and the appropriate treatment could be a challenge for the practitioners, and implies a multidisciplinary approach.

We present the case of a 81-year old retired teacher, which complains of a sudden gait disorder with onset in new year eve. Gait became impossible, the patient fell severe unsteadiness, he can walk only with the aid of 2 persons, and drag his right leg. A fall is reported with a right ankle strain and after this episode, the patient refuses to walk.

His medical history include diabetes mellitus, arterial hypertension, hyperuricemia and Alzhemier dementia. The patient is taking metformine, aspirin, allopurinol, candesartan and rivastigmine for his medical conditions.

A previous CT scan was performed 2 years ago for a minor cranio-cerebral tramatism after an accidental fall, finding important cortical atrophy and normal pressure hydrocephalus, but the neurosurgical opinion at that time was for conservative treatment, and did not provide any indication for shunting.

The patient refuses to go at the hospital, and remain at home for the first 24 hours, being monitored by the family. Blood pressure was 130/80 mmHg, glycemia was 145 mg/dl, no motor deficit was noted on the bed plane, the patient can sit without support, eats normally, has no sphincterian incontinence, was conscious, but has confusional episodes in the evening, as he usually has. He sleeps well at night. Swelling

of his right ankle was noted, and the patient complains of pain in both ankles and in the left knee.

Because the gait trouble did not improve, on next day the patient was admitted to the emergency department of the local hospital with stroke suspicion.

The clinical examination reveals at admission BP=140/80 mHg, rhythmic heart rate 80/min, peripheral pulses were present al lower limbs, skin pallor, painful tumefaction at the right ankle, without ecchimosis, .

The neurologic examination reveals a conscient and oriented patient, cranial nerves examination was normal, in the upper limbs no motor deficit was detected, a mild motor deficit of 4/5 BMRC grade is noted at right lower limb, but the osteotendinous reflexes were normal and symmetrical and the plantar responses were in flexion. No coordination troubles and no sensory disturbancies were noted. The patient can stand only with the aid of 2 persons and cannot walk at all.

Biochemical examinations confirm hyperglycemia at 149 mg/dl, elevated values of CK at 608 UI/L, normal values of cholesterol and triglycerides, normal ionogram and elevated values of uric acid at 8,28 mg/dl. Hemogram were normal, and C reactive Protein (CRP) was mildly elevated (1,5 mg/dl).

Emergency cerebral CT scan found no acute ischemic lesions, cortical atrophy with related dilatation of the ventricular system, diffuse ischemia of white matter (leukoaraiozis). Cerebral MRI confirms the small vessel disease with lacunes and diffuse ischemia of the white matter, cortical atrophy with “ex vacquo” hydrocephalus, but no acute ischemic lesions were detected of diffusion / perfusion sequences.

Radiography of the right ankle reveals signs of an old fracture of the internal malleola.

The psychological examination confirms moderate cognitive dysfunction, loss of autonomy (GAFS score of 20, MMSE score of 18/30), an important memory deficit

(level 1 of 5), but with normal executive functions (BREF score of 16/18).

The patient receives aspirin, fraxiparine, metformin, rosuvastatin and rivastigmine.

Three days later, the gait and stance disorder remains unchanged, but right ankle joint and left knee joint became swollen, eritematous and very painful. CRP was elevated at 3.3 mg/dl and uric acid at 10,34 mg/dl. An acute gout attack was suspected and treated with colchicine, allopurinol and high doses of NSAIDs.

After treatment and pain resolution, kinesitherapy was initiated. After 5 days, the patient improves his neurologic condition: he could stand with unilateral support, and begin to walk with unilateral assistance, and no motor deficit in his lower limbs was noted.

The patient and his family has give their consent for publishing his clinical data.

Although this patient had major neurologic conditions which could lead independently to a gait disorder of neurologic cause (cerebral small vessel disease, Alzheimer dementia, hydrocephalus), the actual cause of his gait disorder was a non neurologic condition. Evaluation of the etiology of a gait disorder should include besides a complete neurological examination, an osteo-articular and metabolic evaluation.

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