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Original article

3D gait analysis in patients with hereditary spastic paraparesis and spastic diplegia: A kinematic, kinetic and EMG comparison

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ABSTRACT

The predominant clinical feature of patients with Hereditary Spastic Paraparesis (HSP) is gait disturbance owing to spasticity and weakness of the lower limbs; the spasticity in early-onset disease (infancy or childhood) often cannot be distinguished from mild form of spastic diplegia (SD). The aim of this study was to quantify the gait strategy in HSP and SD children, focusing on the differences between groups as concerns functional limitation during gait.

9 HSP and 16 SD children were evaluated using Gait Analysis; kinematic and kinetic parameters and EMG pattern during walking were identified and calculated to compare the two gait strategies. The results revealed that these two pathologies are characterised by different gait strategies. In particular we found that knee joint, in terms of kinematics and kinetics, and rectus femoris pattern represent discriminatory aspects in order to compare and differentiate gait patterns of HSP and SD children.

The findings strongly support the issue that HSP and SD patients need individualised therapeutical program, either neurosurgical or pharmacological treatment, based on the quantification of gait deficiencies and in order to address the peculiarity of their motor limitations and to prevent the onset of compensatory strategies.

1. Introduction

Hereditary Spastic Paraparesis (HSP) is a heterogeneous group of neurodegenerative disorders in which the predominant clinical feature is gait disturbance owing to spasticity and weakness of the lower limbs. The spasticity in early-onset disease (infancy or childhood) often does not progress significantly and cannot be distinguished from mild form of spastic diplegia secondary to Cerebral Palsy (CP), except when a family history is elicited.[1,2]

Up to 25% of affected patients are asymptomatic, and so it is possible a patient has only subclinical manifestations,

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therefore escaping diagnosis. The gait abnormalities observed in children with HSP are the earliest clinical finding because classic features for spastic paraplegia are not sometimes seen in these patients;[2] for this reason in these patients a careful and complete clinical evaluation, including gait evaluation, is crucial.

Controversially, there is a lack of detailed quantitative analyses of gait in these patients. Most of the evaluations present in literature were conducted using clinical-functional scales and kinematics of main lower limb joints; in addition they were focused mainly on adult patients.[3–5] As for children with HSP, only one quantitative study investigating the biomechanical strategy during gait is available to our knowledge.

Cimolin et al.[6] quantified the biomechanical strategy in HSP children during gait, comparing their pattern to children who have a mild form of spastic diplegia secondary to CP, as paediatric patients with HSP often resemble diplegic children. Children with HSP, were compared to children with spastic diplegia using Gait Analysis (GA). The main results of this study were the following:

a) HSP and SD were found similar in spatio-temporal and kinematic parameters at proximal joints.
b) The most significant differences were at knee and ankle joints.
c) They both showed tendency to knee hyperextension in midstance, but hyperextension timing was longer in HSP.

In this study, however, the evaluation of gait strategy was focused only on spatio-temporal parameters and kinematic and scanty information (only about ankle power) were given in respect of kinetics and EMG pattern during gait. In addition, a heterogeneous group of spastic paraparesis patients were selected, without considering the genetic origin. From a clinical perspective it is very important to evaluate the gait pattern in these patients using both clinical scores and instrumental quantitative measures, not only with regard to kinematics but also kinetic and EMG data of mainly lower limb joints. The assessment of gait pattern using only spatio-temporal parameters and kinematics is not enough because they give a limited evaluation of patient's walking ability; for this reason the integration of these data with kinetics and EMG is useful for better investigating the joint reactions, moments, powers and muscular activity. In this way it is possible to assess the mechanisms that either control or produce movement, thus potentially developing a more comprehensive understanding of motion and providing insight not only into the 'how' (kinematics), but also into the 'why' (kinetics) of the movement we observe. In addition, dynamic EMG provides the timing and action of muscles that are the prime movers of body segments and bones. Understanding the activity of the muscle as well as the other forces acting on a moving body is critical to understand the root causes of a gait abnormality.

In this context, starting from previous results, the aims of this study are: (a) to quantify the functional limitation of children with genetically defined HSP, using 3D Gait Analysis (GA), in terms of spatio-temporal parameters, kinematics, kinetics and EMG; (b) to identify and calculate the differences between genetically defined HSP and spastic diplegia (SD) secondary to CP, using quantitative parameters obtained from GA data (kinematic, kinetic and EMG data).

2. Materials and methods

2.1. *Subjects*

9 patients with the clinical diagnosis of Hereditary Spastic Paraplegia (HSP; age: 8.9 ± 3.1 years; height: 129.9 ± 9.9 cm; weight: 36.1 ± 8.9 Kg) and 16 patients with Spastic Diplegia (SD; age: 11.9 ± 2.4 years; height: 125.7 ± 7.3 cm; weight: 30.1 ± 7.6 Kg), secondary to Cerebral Palsy, were evaluated in this study.

We studied a referral cohort of HSP patients, according to the following selection criteria: a clinical diagnosis of spastic paraplegia in absence of structural/spinal cord/cerebral disorders, demyelinating, metabolic or inflammatory disorders (particularly for the sporadic forms) and, for the familial forms presence of a positive family history of "gait disturbances" and/or gene mutations (most commonly in the SPG4 and SPG7 genes). All patients underwent metabolic screening; they all had a brain and a spinal cord MRI, reported as normal. All patients have been genetically defined: 4 with mutation in the mitofusin gene, 3 with a mutation in the atlastin gene (inherited by the father) and 2 with a mutation in the spastin gene (a "de novo" mutation, being the parents negative for any mutation in the spastin gene).

Selection criteria for patients with SD were a physician diagnosis of spastic diplegia of Type III according to Rodda's classification,[7] with a mild spasticity of lower limbs joints, no history of cardiovascular disease and no previous surgery or other significant treatments for spasticity.

All patients were able to walk independently without the use of crutches, walkers or braces.

A control group of 15 non-affected subjects (CG; age: 9.2 ± 5.7 years; height: 130.3 ± 7.1 cm; weight: 33.5 ± 9.4 Kg) was included. Selection criteria for this second group included no prior history of cardiovascular, neurological or musculoskeletal disorders. They exhibited normal ROM and muscle strength, and had no apparent postural and motor deficits.

All subjects were volunteers and their parents gave their written consent to the children's participation in this research, in accordance with the local ethical committee requirements.

2.2. *Data collection*

The complete evaluation consisted of three components: clinical examination, video-recording and 3D Gait Analysis (GA).

The "Ashworth scale of muscle spasticity" (ASH) and the Gross Motor Function Measure (GMFM) were assessed in the clinical examination.

The ASH scale evaluates the severity of spasticity calculated as the mean value of spasticity in both lower limbs.[8]

The GMFM measures the child's overall functional abilities and it consists of 88 items, divided into the following sections: 1) lying and rolling; 2) sitting; 3) crawling and kneeling; 4)