



Comparison of temporal and stride characteristics in myotonic dystrophies type 1 and 2 during dual-task walking



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ABSTRACT

Objective: We analyzed temporal and stride characteristics in patients with myotonic dystrophy type 1 (DM1) and type 2 (DM2) while performing dual mental and motor tasks, and investigated correlations between gait parameters and cognitive impairments.

Method: Dual-task walking was performed by 37 patients (20 DM1 and 17 DM2) and 48 healthy subjects divided into two groups, age- and gender-matched control group for DM1 (HC1) and age- and gender-matched control group for DM2 (HC2). The subjects performed a basic walking task, dual-motor task, dual-mental task, and combined motor and mental task.

Results: DM1 and DM2 patients differed significantly in temporal and stride characteristics compared to HC. Main differences in DM1 were slower gait and shorter stride length, while both DM1 and DM2 patients had a higher degree of variation of the swing time during dual-task gait, a parameter that reflects posture and balance. Impact of the cognitive dual task on gait pattern changes was also observed. Visuospatial ability correlated with gait changes in DM1, while executive functions had stronger influence in DM2 ($p < 0.01$). Both patient groups had leg muscle weakness.

Conclusion: Gait pattern was impaired in both patient groups concerning temporal and stride characteristics. Dual-task walking paradigm may discover mild initial gait changes and could provide early identification of fall risks and predict possible falls in DM patients.

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1. Introduction

Myotonic dystrophies (DM) are autosomal dominant multi-systemic disorders [1]. DM type 1 (DM1) is caused by an expansion of CTG repeats (cytosine-thymine-guanine) within the non-coding 3'-untranslated region of the dystrophin myotonic protein kinase (DMPK) gene, while DM type 2 (DM2) is caused by an expansion of CCTG repeats (cytosine-cytosine-thymine-guanine) in the non-coding region of the CCHC-type zinc finger nucleic acid binding protein (CNBP) gene. The main symptoms of DM are: muscle weakness (mainly distal in DM1 and mainly proximal in DM2), myotonia, eye cataract, cardiac conduction abnormalities, insulin resistance, multiple gastrointestinal symptoms, cognitive and behavioral problems, etc. Generally, almost all of these defects are more pronounced in DM1 [1].

Several previous studies investigated gait in subjects with DM1. The main findings were lower gait velocity, reduced cadence, shorter stride length and increased stance phase, mostly due to the distal muscle weakness [2–6]. Significant paresis of ankle plantar flexors seen in DM1 patients may produce a risk of falling, while weakness of dorsal flexors is responsible for the reduction in gait speed. Secondly, in order to oppose the forces acting on the hip joint due to the distal muscle weakness and to maintain balance, irregular position and motion of hips also appear [2,5]. The most important consequences of gait impairments in DM1 are increased risk of stumbles and falls that progresses over time, and causes potential injuries, reduced confidence, activity avoidance, deconditioning, depression, and lower quality of life [3–7].

It was suggested that other symptoms, besides muscle weakness, may potentially contribute to the gait impairments in DM1, including myotonia in leg muscles, visual and hearing impairments, cardiorespiratory dysfunction, obesity, fatigue, and dysfunction of the peripheral and the central nervous system [2–4,6]. To the best of our knowledge, influence of the cognitive

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impairment on gait has not been investigated in DM1 patients so far. Furthermore, gait impairments have not been analyzed in DM2 patients at all and our study is the first report on this topic.

We hypothesize that muscle weakness (more distal in DM1 and more proximal in DM2) significantly affects patients' gait. Gait has been seen as a complex motor behavior, with prominent and varied influence of mental processes, such as executive function or attention [8,9], suggesting that cognitive control is required to compensate for gait deficits. Also, the most important cognitive impairments in these conditions, such as executive dysfunction and visuospatial impairment, may have additional impact on temporal and stride characteristics. We expect gait impairments to be more severe in patients with DM1 since both muscle weakness and cognitive impairments are more pronounced in this form of the disease [1].

The aim of this study was to analyze temporal and stride variability during gait and possible impairments in patients with DM1 and DM2 while performing demanding dual cognitive, motor and combined tasks, investigating the correlation of gait parameters to muscle weakness and possible cognitive impairments.

2. Patients and methods

2.1. Patients

Adult-onset DM1 and DM2 patients were consecutively recruited from the Outpatient unit of the Neurology Clinic from January until December 2014. Forty-eight healthy subjects were tested at the same period on the same gait equipment and were used as control groups – control group of 24 subjects for DM1 patients (HC1) and control group of 24 subjects for DM2 patients (HC2). Statistical analysis showed no significant differences between DM patients groups and their respective control subjects in mean age and gender (Table 1). Patients and control subjects suffering from another condition that could interfere with motor activity (other neurological, psychiatric, somatic, or orthopedic diseases) were excluded. Approval was received from the Ethics Committee of the School of Medicine, University of Belgrade, and written informed consent was obtained from all participating subjects.

Clinical and electrophysiological diagnosis was confirmed by Repeat Primed polymerase chain reaction (RP-PCR) in DM2 patients, and by RP-PCR and Southern blot with repeats count in DM1 [10]. Since the Muscular Impairment Rating Scale (MIRS) [11] is not applicable in DM2, we analyzed the strength of the weakest muscle of the proximal leg and distal leg muscles in both DM1 and DM2 patients (manual muscle testing using 0 to 5 Medical Research Council - MRC scale). The following muscle groups were tested in lying posture: hip flexors, extensors, abductors and adductors, knee flexors and extensors, plantar and dorsal ankle, and toe flexors. Examinations were performed by two independent examiners, specialists in neuromuscular disorders (V.R.S. and S.P) in the morning before cognitive examination. If there was a disparity in the findings, consensus conclusion was reached.

For each patient, cognitive testing was performed by an experienced neuropsychologist after neurological examination and breakfast. The testing lasted for 45 min to 1 h. One to two hours pause was made after cognitive examination and before walking tests. Global cognitive status was assessed using the Mini Mental State Examination (MMSE) with values below 24 meaning cognitive impairment [12]. Copy of the Rey-Osterrieth Complex Figure (ROCF) was used to assess visuospatial and visuoconstructive abilities, where higher scores mean better achievement [13]. Attention was assessed using the Trail Making Test A (TMT-A), while executive functions were examined by the achieved categories on Wisconsin Card Sorting Test (WCST) and the Trail Making Test B (TMT-B) – in all these tests higher scores mean better achievement [13].

2.2. Experimental protocol

Gait performance was measured with and without performing concurrent cognitive and motor tasks (dual-task methodology) [14]. Patients and subjects performed a self-paced basic walking task, a dual-motor task, a dual-mental task, and a combined motor and mental task while walking.

Measurements were performed using GAITRite electronic walkway of 5.5 m active area (CIR Systems, Havertown, PA). Participants performed six passes, three times down the corridor and back, at their comfortable gait velocity, starting and ending their walks approximately 1–1.5 m before and after the walkway

Table 1
Clinical data of DM1 and DM2 patients and two groups of healthy control subjects (HC1 and HC2, respectively).

	DM1 (n=20)	HC1 (n=24)	DM1 vs. HC1	DM2 (n=17)	HC2 (n=24)	DM2 vs. HC2	DM1 vs. DM2
Gender (% females)	40.0	41.7	$\chi^2=0.01$ $p=0.91$	29.4	29.2	$\chi^2=0.00$ $p=0.99$	$\chi^2=0.45$ $p=0.50$
Age (mean years \pm SD)	38.6 \pm 10.9	43.2 \pm 8.9	$t=-1.62$ $p=0.11$	50.7 \pm 8.8	50.1 \pm 10.1	$t=+0.19$ $p=0.85$	$t=-3.68$ $p=0.00$
Disease duration (mean years \pm SD)	14.5 \pm 9.8	-	-	15.8 \pm 13.3	-	-	$p=1.00$
MRC							
Proximal LE	5 (4, 5)			4 (3, 5)			$p=0.03$
Hip flexors	4.5 (4, 5)			4 (3, 5)			
Hip extensors	5 (4, 5)			5 (4, 5)			
Hip abductors	5 (4, 5)			5 (4, 5)			
Hip adductors	4.5 (4, 5)			4 (3, 5)			
Knee flexors	5 (4, 5)			5 (4, 5)			
Knee extensors	4.5 (4, 5)			4.5 (3, 5)			
Distal LE	3.5 (2, 5)			5 (4, 5)			$p=0.00$
Plantar ankle flexors	4 (3, 5)			5 (4, 5)			
Dorsal ankle flexors	3.5 (2, 4)			5 (3, 5)			
Dorsal toe flexors	3 (2, 4)			4 (3, 5)			
Overall MRC score	16.5 (13, 20)			17 (15, 20)			$p=0.84$
CTG repeats (mean \pm SD)	722.4 \pm 259.9	-	-	-	-	-	-

MRC results are presented as median (minimum, maximum);

χ^2 and p values are given for chi square test, t and p values for Student's t -test and p value for Mann-Whitney U test

MRC: Medical Research Council scale; UE: upper extremities; LE: lower extremities.

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