

# Characteristics of two distinct clinical phenotypes of functional (psychogenic) dystonia: follow-up study

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Received: 2 September 2017 / Revised: 4 November 2017 / Accepted: 6 November 2017 / Published online: 15 November 2017  
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## Abstract

**Background** The fixed dystonia phenotype was originally established as a prototype of functional dystonia. Nevertheless, in recent reports different functional dystonia phenotypes have been recognized with dystonic movement comprising phasic instead of tonic contraction.

**Objectives** To examine clinical characteristic in all patients with dystonia who fulfilled the criteria for functional movement disorders irrespective of phenotype in an attempt to determine parameters of clinical presentations that might impact the disease progression pattern and outcome.

**Methods** Patients presented with dystonia features incompatible with organic disease without other features required for the diagnosis of functional movement disorders were analyzed and prospectively followed-up. The two-step cluster analysis was performed to obtain the subgroups of dystonia phenotypes.

**Results** The two-step cluster analysis extracted two subgroup of patients. Patients of the first cluster (68.8%) presented with “mobile” dystonia (84.9%), of cranial/neck/trunk localization (90.9%), fluctuated clinical course (69.7%), with frequent additional movement or other functional neurological disorders (63.6%) during follow-up. In the second cluster

(31.2%) all of the patients presented with “fixed” dystonia of extremities, and the clinical course was characterized by either the disease progression (60%), or continuous without improvement (26.7%), and rare occurrence of additional functional neurological disorders (13.3%).

**Conclusion** In terms of clinical and demographic features as well as pattern of disease progression there are two clinical phenotypes in patients with functional dystonia. Distinctive features of incongruence and inconstancy are characteristic for “mobile” functional dystonia subgroup of patients.

**Keywords** Functional · Dystonia · Mobile · Follow-up

## Introduction

Functional (psychogenic) dystonia (FD) is the second most common and probably the most challenging functional movement disorder (FMD) [1]. Current diagnostic criteria for FMD rely on the assessment of incongruence and inconsistency in both the history and neurological examination, promoting a positive diagnosis, rather than diagnosis by exclusion [2]. It has been recently suggested that features of incongruence and inconsistency were not the same, but rather specific for various phenotypes of FMD [3].

Originally, a specific phenotype of fixed dystonia was established as a FD prototype [4], typically affecting mainly young women and characterized by a sudden onset of fixed abnormal posture of the affected limb, usually precipitated by a minor peripheral trauma. Abnormal posture was frequently accompanied by an early and severe pain, sometimes fulfilling criteria for the chronic regional pain syndrome (CRPS) [4]. Espay and Lang [3] highlighted a rapid onset, fixed dystonia at rest and variable resistance to passive manipulation and/or distractibility or absence of dystonia

**Electronic supplementary material** The online version of this article (<https://doi.org/10.1007/s00415-017-8667-9>) contains supplementary material, which is available to authorized users.

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when unobserved as the core features of FD. More recently, other forms of FD phenotypes have been recognized presenting as an abnormal posture in the neck, face and jaw [5], paroxysmal disorder [6], or dystonia in elderly [7]. These phenotypes were often characterized by different types of phasic instead of tonic dystonic movement (78.4% of cases) [5], or complete absence of fixed dystonia [7]. Also, additional movement disorders (jerks, tremor and gait disorders) and/or other functional neurological symptoms may occur in FD [4–7]. In some cases placebo-like responses to injections of botulinum toxin [8] or transcranial magnetic stimulation [9] were described.

Due to a paucity of prospective studies, little is known whether and how the aforementioned clinical characteristics, including pattern of dystonic movements (tonic vs. phasic), dystonia distribution (extremities vs. axial), or demographic features (young vs. elderly patients), influence clinical course, response to therapy or placebo, and overall outcome [2, 3, 10–12]. Data from studies comprising patients with FMD, including cases with FD, suggested that overall prognosis of FD was poor, although the pattern of progression varied widely [11, 12]. Therefore, we performed a longitudinal study and cluster analysis of clinical characteristics in patients with FD, irrespective of initial phenotype, in an attempt to identify parameters that might be associated with the pattern of disease progression and outcome. We hypothesized that some of the aforementioned characteristics of dystonic movement, distribution, demographic characteristics and associated movement disorders may have impact on the prognosis and the overall course of the disease.

## Patients and methods

Outpatients with FD were recruited from the Movement Disorder Clinic of the Institute of Neurology (University of Belgrade), between November 2010 and November 2015. Only patients with FD fulfilling the criteria of “clinically established without other features” category proposed by Gupta and Lang [2], currently accounted as “clinically definite” [3], were included in further analyses. Briefly, this category indicated the presence of unequivocal clinical features of FMD incompatible with organic disease without the other features (other false signs, multiple somatizations, obvious psychiatric disturbance, and deliberate slowness) required for diagnosis.

In addition to acute mode of onset and fixed posture of affected body part, other features of inconsistency or incongruence with organic dystonia included: (a) variability in performance of the involuntary movements, including amelioration of dystonia during distraction; (b) unusual age at onset for particular phenotype (e.g., early age at onset of blepharospasm (< 40 years of age), late onset (> 25 years of

age) mobile foot dystonia or non-task-specific mobile hand dystonia; (c) persisting unilateral or asymmetric symptoms (e.g., bilateral but asymmetric or unilateral orbicularis oculi spasm with contralateral frontalis overactivity or lower face dystonia); (d) history of spontaneous amelioration or remission of abnormal movements; (e) severe and early pain; and (f) unexpected response to botulinum toxin injections, and/or suggestions. In addition, we specifically looked for other symptoms/signs of FND such as pseudoseizures, “false” weakness (e.g., positive Hoover’s sign, “drift without pronation” sign and “arm drop” test), nonorganic sensory symptoms, visual symptoms (e.g., tubular visual field, convergence spasm), functional gait disorders (e.g., monoplegic “dragging” gait, “walking on ice” pattern, uneconomic postures with waste of muscle energy) [13–15] and presence of other functional movement disorders (tremor, myoclonus, tics, parkinsonism) [3]. Presence of multiple somatizations and previously diagnosed psychiatric disease were noted, but these features were not discriminatory for the diagnosis of organic vs. functional dystonia. Patients with functional overlay of organic dystonia phenotype were not included.

Recommended examinations for secondary dystonia [16], including brain MRI, were normal in all patients. Wilson’s disease was excluded in all patients under the age of 55 years. Genetic tests for *DYT1* and *DYT6* mutations were negative in all patients, as well as mutations in *DYT11* and *Parkin* gene in cases resembling dystonia–myoclonus phenotype and with late onset foot/hand dystonia, respectively.

The included patients underwent semi-structured interview that assessed precipitating events, disease evolution, treatment responses and clinical course. Severity of dystonic movements was assessed by the Unified Dystonia Rating Scale [17] and the Burke–Fahn–Marsden Dystonia Rating Scale [18]. Global cognitive assessment was performed using the Mini-Mental State Examination (MMSE) [19], while psychiatric evaluation included the Hamilton Depression Rating Scale (HDRS) [20], the Hamilton Anxiety Rating Scale (HARS) [21], the Apathy Scale [22], the Somaform Dissociation Questionnaire (SDQ-20) [23], and the Dissociative Experiences Scale II (DES-II) [24].

In all patients diagnosis and treatment options was explained by one of us. Treatment options included botulinum toxin therapy, cognitive behavioral therapy and physiotherapy, each of was applied only if patients were willing to participate. Patients were regularly followed-up in 4–6 month intervals, when the evaluation included neurological examination and semi-structured interview to comprise disease evolution, treatment response and clinical course. On each visit the outcomes were rated as: (1) better or without symptoms; (2) unchanged; or (3) worse. Disease course (final outcome) was defined as: (1) progressive (worsening or spreading of dystonia with or without appearance of additional FMD or FND, and without periods of improvement); (2) fluctuating

(with improvements/remissions, when subsequent relapses consisted of the same or other FMD); (3) stationary (patients were without improvement or worsening of dystonia, and with no additional FMD or FND). Remissions were defined as complete or almost complete (minor residual symptoms) resolution of symptoms and signs lasting at least 12 months.

Written informed consent was obtained from all patients and the study was approved by the Ethical Committee of the Faculty of Medicine, University of Belgrade.

### Statistical analyses

The descriptive data were presented through means, standard deviations, frequencies, and percentages. The data were analyzed in two phases. First, the two-step cluster analysis was performed to obtain the clusters of patients using five main phenotypic characteristics as predictor variables: the presence of fixed dystonia, the presence of cranial/neck/trunk dystonia localization, the presence of CPRS, the presence of additional movement disorders or FND, and the presence of spontaneous or therapeutic improvement. The number of clusters was obtained in exploratory manner (automatically extracted by the clustering procedure, with no pre-defined number of clusters). The Silhouette measure of separation and cohesion was used to present the cluster quality, indicating good or fair quality if the values are above 0.5 and 0.2, respectively. The importance of predictor variables used for clustering was described on the 0.0–1.0 scale.

In the second phase, the two obtained clusters of patients were analyzed for their additional differences.  $\chi^2$  test or Fisher's exact test were used for categorical variables, whereas *t* test, Mann–Whitney *U* test and Kruskal–Wallis test were used for continuous variables (in respect to the normality of distribution).

### Results

The study comprised 48 FD patients (37 females; 77%) (Table 3 in Appendix). The mean age at onset was  $41.8 \pm 12.7$  years, with almost a third of patients (28.9%) being older than 50 years at onset, and only 6 of them were (12.5%) with the appearance of symptoms before the age of 20 years. A psychiatric disorder was present in 60% of cases prior to the diagnosis of FD (77.8% had received psychiatric treatment, but none reported administration of dopamine receptor blockers), while 70.8% of cases reported previous psychological stressor, although in only half of them clear temporal relationship to the onset of dystonia could be identified. Acute onset of dystonic movements was identified in 42 patients (87.5%). In 18 cases (37.5%) dystonic movements affected extremities, characterized by an early fixed abnormal posture in 15 of them (83.3%). The remaining three cases presented with rest

and/or action-induced intermittent muscle contraction of the extremities, causing abnormal repetitive movements, postures, or both, resembling dystonia (Video 1, Segment 1). No patient reported task-specific dystonia (e.g., writer's cramp). When the leg was affected by abnormal movements presented mainly during walking (Video 2, Segment 1). Thirty cases initially had axial distribution of dystonic movements, with fixed posturing of the neck, lips or eyelids affection in 5 of them (16.7%). No patients reported voluntary control, premonitory urge, or gestes antagonistes maneuver.

The disease course over the mean follow-up duration of  $3.1 \pm 1.4$  years was stationary in 20.8% of cases, progressive in 27.1%, while the remaining 52.1% had clinical course characterized by spontaneous or therapy-induced (placebo like) improvements/remissions followed by subsequent relapses. Only two cases (4.2%) had stable remissions without disease recurrence during follow-ups of 20 and 24 months, respectively. Spreading of dystonia to other body region was observed in 35.4% of cases after the mean time of  $25.5 \pm 31.0$  months. Appearance of additional movement disorders or other functional neurological symptoms was noted in 14 (29.2%) and 16 patients (33.3%), respectively. Tremor (30.3%) and gait disturbances (30.3%) were the most frequent, followed by nonorganic sensory symptoms (18.2%), myoclonus (15.1%), "false" weakness (12.1%), hypokinesia (6.7%) and pseudoseizures (6.2%).

The two-step cluster analysis extracted two clusters of patients: group 1 ( $n = 33$ ; 68.8%) and group 2 ( $n = 15$ ; 31.2%). The ratio of sizes between these two clusters was 2.2, which was adequate since it is generally preferred for the ratio of sizes between the largest and the smallest cluster to be below 3. The Silhouette measure of separation and cohesion was 0.5 (i.e., cluster quality between fair and good). The most important predictor of clustering was axial (cranial/neck/trunk) distribution of dystonic movements (1.0), followed by the presence of fixed dystonia (0.85), presence of CPRS (0.53), and the presence of improvement (0.40), while the least important predictor was the presence of additional movement or functional disorder (0.33).

Considering clinical characteristics of the two identified clusters (Tables 1, 2), patients in group 1 dominantly presented with cranial/neck/trunk dystonia distribution ( $n = 30$ ; 90.9%), fluctuating clinical course ( $n = 23$ ; 69.7%) and appearance of additional movements or other functional neurological disorders ( $n = 21$ ; 63.6%) (Video 1, Video 2; Segment 2). Only 5 of those cases (15.1%) had fixed dystonia at presentation, and none had CPRS. In group 2, all 15 patients presented with fixed dystonia of extremities, and associated CPRS in almost half of them (7 patients; 46.7%). In group 2, clinical course was progressive (9 patients; 60%) (Video 3) or stationary (4 patients; 26.7%). Some improvement/remission was noted in two patients (13.3%), with the same number of patients expressing additional FMD/FND.

**Table 1** Clinical features of 48 FD cases separated into two groups

Characteristics	Group 1	Group 2	<i>p</i> value
Number of patients	33 (68.8%)	15 (31.3%)	–
Current age (years) <sup>c</sup>	52.8 ± 11.9	40.0 ± 14.6	0.007 <sup>a</sup>
Age at onset (years) <sup>c</sup>	45.2 ± 10.4	34.3 ± 14.3	0.022 <sup>a</sup>
Females	26 (78.8%)	11 (73.3%)	0.720 <sup>b</sup>
Education (years) <sup>c</sup>	11.5 ± 1.9	11.0 ± 1.6	0.371 <sup>a</sup>
Disease duration (years) <sup>c</sup>	7.5 ± 5.9	5.5 ± 3.5	0.384 <sup>a</sup>
Acute onset	28 (84.8%)	14 (93.3%)	0.650 <sup>b</sup>
Localisation—extremities	3 (9.1%)	15 (100%)	< 0.001 <sup>b</sup>
Localization—cranial/neck/trunk	30 (90.9%)	0 (0%)	< 0.001 <sup>b</sup>
Fixed dystonia	5 (15.2%)	15 (100%)	< 0.001 <sup>b</sup>
Pain at dystonia localization	19 (57.6%)	15 (100%)	0.002 <sup>b</sup>
CPRS	0 (0%)	7 (46.7%)	< 0.001 <sup>b</sup>
Physical trauma preceding abnormal movements	7 (21.2%)	4 (26.7%)	0.720 <sup>b</sup>
Psychological trauma preceding abnormal movements	25 (75.8%)	9 (60%)	0.315 <sup>b</sup>
Psychiatric disorder before the onset of dystonia	18 (58.1%)	9 (64.3%)	0.948 <sup>b</sup>
Previous psychiatric treatment	13 (41.9%)	9 (64.3%)	0.286 <sup>b</sup>
FMS score <sup>c</sup>	7.2 ± 4.8	19.4 ± 7.7	< 0.001 <sup>a</sup>
UDRS total <sup>c</sup>	8.8 ± 5.6	14.1 ± 7.4	0.007 <sup>a</sup>
MMSE <sup>c</sup>	27.9 ± 1.6	29.0 ± 1.3	0.039 <sup>a</sup>
HAMD <sup>c</sup>	17.1 ± 9.7	14.1 ± 10.2	0.411 <sup>a</sup>
HAMA <sup>c</sup>	14.1 ± 10.1	12.0 ± 10.2	0.447 <sup>a</sup>
Apathy score <sup>c</sup>	19.3 ± 11.3	14.0 ± 10.8	0.134 <sup>a</sup>
DES <sup>c</sup>	3.8 ± 6.4	3.9 ± 7.2	0.774 <sup>a</sup>
QSD <sup>c</sup>	26.9 ± 9.5	30.3 ± 10.1	0.194 <sup>a</sup>
Treatment			
BT	29 (87.9%)	13 (86.7%)	1.000 <sup>b</sup>
CBT	19 (57.6%)	9 (60%)	1.000 <sup>b</sup>
Physiotherapy	17 (51.5%)	13 (86.7%)	0.026 <sup>b</sup>

Values presented as number of patients with percentages in parenthesis or <sup>c</sup> as means ± standard deviations  
*CPRS* chronic regional pain syndrome; *MMSE* Mini-Mental State Examination, *HDRS* Hamilton Depression Rating Scale; *HARS* Hamilton Anxiety Rating Scale; *AS* Apathy scale; *SDQ-20* Somatoform Dissociation Questionnaire; *DES-II* Dissociative Experiences Scale II; *UDRS* Unified Dystonia Rating Scale; *FMS* Burke–Fahn–Marsden Dystonia Rating Scale; *RR* remission/relapses; *CBT* cognitive behavioral therapy, *BT* botulinum toxin

<sup>a</sup>Depending on the parametric or non-parametric properties of the variable, *t* test or Mann–Whitney *U* test was performed

<sup>b</sup> $\chi^2$  test or Fisher's exact test were performed where appropriate

In respect to other phenotypic differences between the two groups (Table 2), patients in group 2 were significantly younger at the disease onset, had higher scores on dystonia scales, associated pain was more common, the MMSE scores were higher, and physiotherapy was more commonly applied. Spreading of dystonic movements was more frequent in group 2 (60%), whereas group 1 was more commonly associated with additional movement disorders (63.3%) (Table 2). Patients with improvements and relapses were more prevalent in group 1, comparing to more stationary course in group 2. No significant differences were found between groups in respect to the scores on psychiatric scales, preceding physical trauma and psychological stressors.

## Discussion

The main finding of this study is that regarding disease progression and overall prognosis two clinically distinctive phenotypes may exist in patients with FD. One (group 2) is characterized by symptom onset in mid-thirties, prominent pain, early fixed abnormal posture mainly affecting extremities (i.e., fixed dystonia), often associated with CPRS. During the mean follow-up of 3 years, in 26.7% of these patients the disorder remained stationary, while 60% had progressive deterioration (mainly spreading of dystonia, with low tendency for development of other FMD or FND). In the second group (group 1) patients presented with rest

**Table 2** Characteristics of disease course in 48 FD patients separated in two groups

Characteristic	Group 1	Group 2	<i>p</i> value
Follow-up (years) <sup>d</sup>	3.1 ± 1.5	3.1 ± 1.2	0.875 <sup>a</sup>
Course of disease			< 0.001 <sup>b</sup>
Stationary (S)	6 (18.2)	4 (26.7)	S vs. I/RR 0.043 <sup>c</sup>
Improvement/RR (I/RR)	23 (69.7)	2 (13.3)	S vs. 0.222 <sup>c</sup>
Progression (P)	4 (12.1)	9 (60.0)	I/RR vs. < 0.001 <sup>c</sup>
Stable remission	2 (6.1%)	0	1.00 <sup>b</sup>
Additional movement/functional disorders	21 (63.6)	2 (13.3)	0.003 <sup>b</sup>
Time to onset of additional movement/functional disorder (months) <sup>d</sup>	24.2 ± 41.0	36.0 ± 16.9	0.285 <sup>a</sup>
Dystonia progression	8 (24.2)	9 (60.0)	0.038 <sup>b</sup>
Time to dystonia progression (months) <sup>d</sup>	34.9 ± 41.5	17.1 ± 15.8	0.481 <sup>a</sup>
Additional movement disorder	13 (39.4)	1 (6.7)	0.037 <sup>b</sup>
Time to onset of additional movement disorder (months) <sup>d</sup>	15.5 ± 20.8	24.0	0.571 <sup>a</sup>

Values presented as number of patients with percentages in parenthesis or <sup>d</sup> as means ± standard deviations

<sup>a</sup>Depending on the parametric or non-parametric properties of the variable, *t* test or Mann–Whitney *U* test was performed

<sup>b</sup> $\chi^2$  test or Fisher's exact test were performed where appropriate

<sup>c</sup>Bonferroni significance correction 0.05/3 = 0.017 (post hoc Fisher's exact tests)

or action-induced intermittent muscle contraction causing abnormal movements and postures, mainly but not exclusively with cranial and neck distribution. In majority of them clinical course was characterized by improvements or remissions, followed by subsequent relapses with recurrence of dystonic movements and appearance of additional movement disorders, but with less tendency to dystonia spreading. Both groups had rapid onset of dystonic movements, female predominance, and high prevalence of psychological and physical stressors (Tables 1, 2).

Clinical characteristics in group 2 were similar to those originally described by Schrag et al. [4] (“fixed” dystonia phenotype), whereas the phasic contractions in group 1 without “fixed” postures more closely resembled “organic” dystonia (“mobile” dystonia phenotype) [25]. The characteristics of “mobile” subgroup were also similar to recently described phenotypes of FD affecting the face [5] and FMD in the elderly [7]. Fasano et al. [5] reported that only 27.5% of their 51 cases with facial FD had “fixed” dystonic posture, while in a series of Batla et al. [7]. None of nine FD cases with symptoms onset after 60 years of age had “fixed” dystonia phenotype. Accordingly, our patients in “mobile” subgroup had predominant cranial and neck affection and were on average 10 years older than those with “fixed” dystonia. Moreover, 10 of our 11 cases with dystonia onset after the age of 50 years belong to the “mobile” subgroup, confirming that dystonia may be a presenting feature of FMD in older adults and elderly patients, but with clinical expression other than the “fixed” dystonia phenotype. In absence of the fixed postures and prominent pain of affected regions, features of inconsistency or incongruence

with organic dystonia in “mobile” subgroup included rapid onset of abnormal movements [4], amelioration of dystonic movements during distraction or suggestions [26], unusual age of onset for a particular phenotype [1], history of spontaneous remission [27] and unusual (immediate) response to botulinum toxin injections [8]. For example, 69.7% of cases in “mobile” subgroup had fluctuating (inconsistent) clinical course with spontaneous or placebo-like therapy-induced improvements, but subsequent relapses. Moreover, in 63.3% of these cases besides dystonia, relapses were manifested with other functional neurological signs. In continuation to previous finding in a group of elderly FMD patients [7], functional gait disturbances were the most prevalent additional symptom in “mobile” FD subgroup. In a third of cases from this group, extremities were affected in isolation (four cases) or combined with cranial/neck affection. Therefore, in addition to recent description of phasic contraction affecting face [5] our results suggest that FD of extremities may also present as “mobile” dystonia, leading to intermittent abnormal movements and postures. During the follow-up period, none of patients from “mobile” subgroup developed characteristics of “fixed” dystonia phenotype, whereas two cases presented with “fixed” abnormal posture converted to “mobile” subgroup.

In both groups, we found a high prevalence of psychiatric comorbidity, in line with previous studies [10, 11, 28]. Two groups did not differ in terms of presence of negative life events in the year prior to symptom onset and scores on neuropsychiatric scales (Table 1). The lack of differences between the groups may be due to a small sample size, insufficient sensitivity of applied instruments,

but also due to a common underlying psychopathology. Moreover, recent data failed to find specific psychiatric background in FMD patients (including FD) when compared to patients with organic disease and healthy controls [29, 30], indicating that they are not universal or mandatory for diagnosis. Nevertheless, actual framework of FMD still includes a potential role of the ‘presumed psychological factors’, but since we have not performed structured psychiatric interview, we cannot exclude possible differences of categorical psychiatric diagnoses between the identified groups. Considering recent description of various neurobiological abnormalities in FMD patients, such as decreased interoceptive sensitivity [31], alexithymia [32], loss of sensory attenuation [33], and impaired abstract probabilistic reasoning [34], it would be interesting to explore whether specific clinical FD subgroups have a common underlying neurobiology or certain differences nevertheless are present.

The major limitations of our study included a relatively small number of patients, unblinded rating, referral bias inherent for a tertiary center, and the fact that only cases fulfilling the criteria for “clinically definite” were analyzed (therefore, we could not rule out the presence of other phenotypes of FD in the community). For example, none of our FD patients presented with characteristics of recently described paroxysmal FMD [6]. Second, although the same multidisciplinary therapy was offered to all our patients, they were not treated in the uniform way and our results should not be interpreted to exclude or confirm the influence of specific treatments modalities on disease course and outcome. For example, 86.7% of patients with “fixed” dystonia were treated by physiotherapy, but in only few of them we were able to offer specific physiotherapy procedures according to recent consensus recommendations [35]. In addition, we cannot exclude the potential bias (placebo effect) that might be present when there is heterogeneous treatment allocation. Finally, the two-step cluster analysis yielded two phenotypic clusters of fair-to-good quality, leaving the space for future exploration of additional specific clustering characteristics in a larger sample. Further studies on a larger sample of patients with FD and with a longer period of prospective follow-up are needed to confirm existence of proposed subgroups as well as their distinct pattern of disease progression.

In conclusion, results from this study confirm previous findings that FD may present with “fixed” and “mobile” phenotype, and suggest phenotype-specific heterogeneity of disease progression and overall prognosis. “Mobile” FD affecting extremities may be under recognized phenotype of FD and careful identification of distinctive features of incongruence and inconsistency is necessary for early identification of these cases.

## Compliance with ethical standards

**Financial disclosure** This study was supported by the Ministry of Education and Science of the Republic of Serbia (Grant no. 175090).

**Conflicts of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

**Ethical standards** All persons gave their informed consent prior to their inclusion in the study. The manuscript does not contain clinical studies or patient data.

## Appendix

See Table 3.

**Table 3** Demographic and clinical features of 48 FD patients fulfilling criteria for FD

Characteristic	
Current age (years) <sup>a</sup>	48.8 ± 14.1
Age at onset (years) <sup>a</sup>	41.8 ± 12.7
Disease duration (years) <sup>a</sup>	6.9 ± 5.3
Follow-up (years) <sup>a</sup>	3.1 ± 1.4
Acute onset	42 (87.5%)
Localization—extremities	18 (37.5%)
Localization—cranial/neck/trunk	30 (62.5%)
Fixed dystonia	20 (41.7%)
Pain at dystonia localization	34 (70.8%)
CPRS	7 (14.6%)
Physical trauma preceding abnormal movements	11 (22.9%)
Psychological trauma preceding abnormal movements	34 (70.8%)
Psychiatric disorder before the onset of dystonia	27 (60.0%)
Previous psychiatric treatment	22 (48.9%)
FMS score <sup>a</sup>	11.0 ± 8.1
UDRS total <sup>a</sup>	10.5 ± 6.6
MMSE <sup>a</sup>	28.3 ± 1.6
HAMD <sup>a</sup>	16.2 ± 9.9
HAMA <sup>a</sup>	13.4 ± 10.1
Apathy scale <sup>a</sup>	17.7 ± 11.3
DES <sup>a</sup>	3.8 ± 6.6
QSD <sup>a</sup>	28.0 ± 9.7
Course of illness	
Stationary	10 (20.8%)
Improvement/RR	25 (52.1%)
Progression	13 (27.1%)
Additional movement disorder	14 (29.2%)
Additional functional disorder	16 (33.3%)

Values presented as number of patients with percentages in parenthesis or <sup>a</sup> as means ± standard deviations

CPRS chronic regional pain syndrome, MMSE Mini-Mental State Examination, HDRS Hamilton Depression Rating Scale, HARS Hamilton Anxiety Rating Scale, AS Apathy scale, SDQ-20 Somatoform Dissociation Questionnaire, DES-II Dissociative Experiences Scale II, UDRS Unified Dystonia Rating Scale, FMS Burke–Fahn–Marsden Dystonia Rating Scale, RR remission/relapses

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