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# Functional Movement Disorder and Parkinson's Disease Comorbidity: A Case Report

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**SUMMARY**

Functional movement disorders (FMD) are abnormal involuntary movements that are not attributable to known neurological and neuroanatomical causes. In the past decade, FMD diagnoses have been based on positive factors that are inconsistent with neurological disorders and not on the exclusion of neurological disorders. In this report, we presented the case of a female patient who had been followed up for 4 years in multiple health centers with the diagnosis of a neurological disorder which was suspected in the previous 1 year to be of psychogenic origin. A neurological disorder comorbid with depression and FMD was diagnosed after admission as an inpatient to our clinic. By presenting this case, it was intended to emphasise the importance of follow-up after diagnosing FMD, given its common basis with Parkinson's disease and the high incidence of comorbidites seen with it. Imaging and elecrophysiological techniques should be relied upon for differentiating FMD and neurological diseases. Although psychological causes are significant disease risk and/or maintenance factors, they are not sufficient for explaining the aetiology of FMD, which requires a multidisciplinary approach.

**Keywords:** Functional movement disorders, parkinson's disease, DaTscan

## INTRODUCTION

Functional movement disorders (FMD), first described as "hystera" by the Greeks, are abnormal and involuntary movements not attributable to neurological or neuroanatomical causes and were explained by Freud with the hypothetical term "conversion", suggesting the transformation of unconscious intrapsychic conflicts to neurological physical symptoms (Ford and Folks 1985). The term "psychogenic" was commonly used upon realisation of the concomitant psychological factors (Morgante et al. 2013). The new term "functional disorder" has recently been used in being less stigmatising and more patient-friendly (Edwards et al. 2014). In the past decade, FMD diagnosis has been based on positive factors that are not consistent with known neurological disorders rather than by the exclusion of the possible neurological disorders (Carson and Lehn 2017).

In the Diagnostic and Statistical Manual of Mental Disorders fourth edition (DSM-IV), the diagnostic criterion A defines Conversion Disorder (CD) as the relationship between a functional, medically unexplained neurological symptom, and the criterion B gives psychological stressor as the basis (American Psychiatric Association-APA, 2000). The DSM-5 (APA 2013) stresses that CD should be diagnosed on the basis of the "positive" clinical findings and by less weighting of the associated psychological factors. The new criterion B states that "evidence is necessary for showing the incompatibility between the clinical findings and the recognized neurological or medical conditions" (Stone et al. 2011a). The evidence for the "incompatibility" of the patient's symptoms with the functional neurological disorder is obtained through specific and convenient neurological examination (Stone et al. 2011b) or electroencephalography (EEG), electromyography (EMG) and Striatal dopamine transporter scan (DaTscan) (Kuris and Pareés 2020). Earlier research reports associated CD especially with social and occupational life events, emotional stress, maltreatment and early childhood traumas (Widiger and Mullins-Sweatt 2005). It has been emphasized in the DSM-5 classification that FMD diagnosis could be made without any recognized psychological triggering factor (Stone

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and Edward 2011, Edwards and Bhatia 2012). Although Kranick et al. (2011) reported higher incidences of childhood trauma in FMD patients as compared to healthy volunteers, psychopathology was not always clear. A significant majority of patients with FMD did not report any traumatic event in their past (Roelofs and Pasman 2017). Even though psychological factors alone were insufficient to explain the aetiology of FMD, these were said to be significant risk and maintenance factors (Stone and Edward 2011, Edwards and Bhatia 2012). In the literature, there has been a general shift from psychogenic movement disorders to FMD, resulting in more emphasis on a biopsychosocial disorder model comprising multiple symptom causing factors (Pick et al. 2019).

FMD cases with significant disabilities have been reported to be frequently seen in neurology clinics (Carson and Lehn 2016). Although FMD is widely recognized (Hallett 2006), there is debate on the qualifications of the physician who is to follow up the patients and manage their therapy (Hallett 2019, Perez et al. 2019). There has been significant progress in the identification of the factors enabling the recognition of FMD as a neurobiological disorder at the junction of neurology and psychiatry (Espay et al. 2018, Baizabal-Carvall et al. 2019). Indeed, evidence was obtained to suggest abnormal emotional regulation in FMD with increased activity in the amygdala and periaqueductal grey matter during adverse psysiopathological emotional stimulation (Voonet al. 2010). Lack of habituation in the amygdala activity was suggestive of a general hyperarousal in FMD. In comparison to healthy controls, these patients were reported to be more prone to automatic motor defense behaviours such as the freeze response mediated by the periaqueductal grey matter demonstrated to play a significant role in the freeze response in experiments carried out with both animals (Koutsikou et al. 2015) and humans (Hermans et al. 2013, Blakemore et al. 2016). Assessment of FMD in adolescents and children through their heart rates indicated reduced parasympathetic response to sympathetic stressors (Kozlowska et al. 2015). The DSM-5 classification has advantages over that of the DSM-IV by not only pinning the term FMD, but also in identifying its subtypes according to symptoms including dystonia, torticollis, facial movement disorders, tremor, gait disorders, parkinsonism, myoclonus, and tics (Barbey and Aybek 2017). As the most frequently reported psychogenic movement disorders, dystonia and tremor appear in 10% of the FMD cases with symptoms of parkinsonism (Barbey and Aybek 2017).

This report discusses the case of a female patient followed up during the previous 1 year with suspected psychogenic movement disorder after a 3-year follow up for a neurological disorder; and finally diagnosed in our clinic with FMD comorbid with a neurological disorder.

## CASE

The 42-year-old married female patient, whose details of identity have been changed with her written consent for this report, consulted the emergency services with complaints of difficulty of walking over the previous 4 years, imbalance, fatigue, depressed mood, anhedonia, and suicidal ideation. She was admitted to the psychiatry unit as an inpatient for differential diagnosis.

It was learned that the patient, who had been complaining of gait difficulty, clumsiness, imbalance, limping and reduced movement on the right side since 2015, had been initially diagnosed with Parkinson's Disease (PD) and put on pramipexole treatment for one year. She had also been prescribed levodopa which did not result in significant improvement in her complaints. The patient explained that in the clinic she previously consulted, her complaints were found to be of psychogenic origin, and she was directed to a psychiatry clinic. At the psychiatry clinic she had consulted 3 years previously with the same complaints, she was prescribed venlafaxine which she used irregularly without improvements in her depression and gait difficulty. On account of weakness and shaking, especially on the right side, and spilling food when eating, she tended over the previous 6 months to weep in depressed mood, had anhedonia, difficulty in falling asleep. Losing her functionality, failing to keep up with childcare and house chores compounded by marital problems, she attempted to commit suicide by drug taking 3 days before consulting the hospital. The attempt, which she stated she did not regret, was prevented by her husband.

It was determined that the patient had normal motor and mental development, she did not have a history of cranial trauma or seizures, alcohol-cigarette or substance use, a family history of psychiatric disorder, or family relationships between her parents. Her maternal grandmother and her father's uncle had been diagnosed with Parkinson's Disease (PD). It was also learned that the patient had extremely traumatic early life experiences, having been married off at the age of 17 to a man of her own age, which she had accepted in order to escape her father's violence, which had been "destructive" and resulted in divorce, separating her from her children when she did not receive enough family support.

Mental state examination: The patient looked her age, appeared to have moderate level of self care, was cooperative and fully oriented with normal attention, natural pace and volume of speech, the normal ability of reality testing and judgment. Her mood and affect were depressive, tending to tearfulness. Her thought contents included feelings of guilt, despair and suicidal ideation in believing her disability to be a punishment and would not improve. She did not want to live anymore.

At the start of admission to the clinic her scores on the Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI) were 48 and 17, respectively. Venlafaxine 75 mg/day and diazepam 2*5 mg were administered for her depressive symptoms and daily supportive interviews were carried out throughout her hospital stay. The Minnesota Multiphasic Personality Inventory (MMPI) indicated that the patient somaticized her psychological problems, showing physical symptoms under stress and using projection defense mechanisms such as repression, denial, and rationalization. She avoided facing her emotions, rarely showed anger, did not express any negative emotion and demonstrated passive-aggressive behaviours.

During her stay at our clinic, it was noted that the distance she could walk with support was shortened to 10 steps and that she could go to the lavatory with the help of her relations. Her gait pattern involving difficulty in starting movements, rigidity of the extremities, walking with small, dragged steps necessitated eliminating neurological pathology. Therefore, the neurology unit was consulted. Her neurological examination confirmed that she was conscious, cooperative, and oriented. Her muscles had full strength; the lower extremity deep tendon reflexes (DTR) were normal; pathological reflexes or fasciculation were not detected; she had hypomimia and bilateral rigidity more evident on the right. The patient was anxious and cautious when walking with a bent-over posture, small steps, without ankle flexibility, and arm swing which, however, was normalised when running. She could sit only with assistance. EMG was reported as "normal". Cranial Magnetic Resonance Imaging (MRI) was within "normal limits" but MRI of the spine showed "spinal dischopathies". Routine biochemical blood test results were within normal limits. Wilson's Disease was eliminated by normal results on blood caeruloplasmin and copper levels and the 24-hour urinary copper excretion.

When the neurology department reevaluated the patient on the 15th day of her hospitalization, bilateral rigidity was confirmed with hypomimia more evident on the right. Her walk was slow but indicated an improvement and rotations and motions of sitting and standing were better. She described the occurrence of leg cramps from time to time. However, fasciculation was not observed. The neurology department suggested that the patient had FMD.

During the follow up of her psychiatric complaints, venlafaxine dose was titrated to 225 mg/day, resulting in improvements of the depression symptoms and sleep pattern and disappearance of suicidal ideation. Her BDI and BAI scores were 19 and 12, respectively. Despite the improvement of her balance, the rigidity and the cautious, slow gait persisted. Upon her request, due to her son's problems, the patient was discharged with the recommendation of weekly follow-up appointments.

In the 3rd week follow-up visit after her discharge, the BDI and BAI scores were 17 and 12, respectively. The patient described improvement in the relationships with her husband and children. She had started seeing her children from her former spouse, and her depression had significantly improved. Neurological examination showed bilateral rigidity and bradykinesia. She was still walking with slow, short steps and a bend-over posture and was worried about the worsening of her neurological complaints.

In the 4th-week follow-up visit, her gait pattern and neurological complaints were the same. On grounds of functional complaints disproportinate with the results of her neurological examinations, Spect Brain DaTscan was carried out with the recommendation of the neurology unit, which demonstrated the asymmetric loss in the nigrostriatal system innervation and the consistence of the imaging obtained with PD. The patient was started on levodopa + benserazide (100mg+25mg) 3*1. After one week of treatment, the patient's arm motions and step length increased, and gait posture improved. Her depressive complaints significantly decreased, and her BDI and BAI scores were 12 and 7, respectively. The patient is still being followed up in psychiatry and neurology clinics.

## DISCUSSION

Incompatibility of the patient's history with neuroanatomical and neurological causes and observation of distinct changes in symptoms with continuous spontaneous remissions can suggest a functional basis in cases of FMD (Gasca-Salas and Lang 2017). Observation of changes in the neurological examination results with the given psychiatric therapy supported the FMD diagnosis in our patient. In functional parkinsonism tremor usually affects the dominant hand. Whereas the resting tremor typically decreases with motion in PD, it mostly occurs equally during resting, standing, and moving in FMD. Another difference is the reemergent tremor which, classically seen in PD, is "a short pause of tremor during upper limb posture" (Jankovic et al. 1999). Tremor in FMD decreases during walking and with distraction in contrast to the resting tremor in PD. However, tremor was not observed in our patient.

Although repetitive motions might be slow in functional PD (fPD), true bradykinesia with a reduction in amplitude or stiffness was not reported (Morgan et al. 2004). In psychogenic PD, however, an active resistance without cogwheel was reported during the assessment of muscle tone which, in contrast to functional PD, subsided after distraction maneuvers (Lang et al. 1995). Examination showed increased muscle tone in our patient. Bradykinesia, especially more evident in the upper extremity, was a significant symptom in our patient indicating an additional pathology. Generalised

abnormal gait has been reported in FMD, with incidences of 5.7% and 40%, respectively, of isolated gait disorders and those concurrent with other movement disorders (Baikand Lang 2007). 'Knee-buckling' was the most common pure functional gait model, followed by astasia-abasia (Barbey and Aybek 2017). Exaggerated compensatory reactions such as spreading arms as in tightrope walking and significantly short step heights and lengths as in walking on ice have been reported in FMD (Baik and Lee 2012). Our patient also had reduced step heights and lengths characterized by ''walking on ice" together with impaired balance. The efforts of balancing were accompanied by exaggerated arm movements. Her gait was hesitant and slow. This kind of atypical gait pattern was a significant symptom that suggested the diagnosis of FMD. The types of gait with 94-100% specifity for FMD have been ranked as (1) momentary fluctuations of stance and gait, (2) excessive slowness or hesitation, (3) "psychogenic" Romberg test with increasing sway amplitudes improved by distraction, (4) the "walking on ice" gait pattern and (6) sudden buckling of the knees and (7) hemiparetic walking with foot-dragging in every stage of the gait. "Huffing and puffing" signs involving anger, grimace, and breath-holding were reported in 44% of FMD patients (Lempert et al. 1991), which were either seen minimally or not at all, with a 89% -100% specificity to cases of organic gait disorder (Laubet al. 2015). Mental distraction can improve FMD rather than worsening it. Patients were observed to complain about impaired balance or falling while displaying good balance control for stabilization with ability to make exaggerated body movements or "astasia-abasia". Tremor was decreased with distraction and increased by focusing the attention; and bradykinesia occurred in almost all patients with an atypical slow course (Moene et al. 2002). Arm movements improved during running (Kuris and Pareés 2020).

Our patient had all the signs of atypic gait pattern compatible with the positive diagnostic criteria for FMD by making much effort when standing up with distressful facial movements or "huffing-puffing", walking with excessive slowness and caution, having recurrent episodes of falling in the ward, and feeling off-balance, changing her posture to maintain balance and by foot-dragging or the hemiparetic gait and taking short steps as if "walking on ice". The slow start of the patient's symptoms, the presence of hypomimia, distinct and unilateral bradykinesia, and rigidity were considered as indicative of PD; while becoming active enough to run after starting to move with much difficulty and the improvement of arm movements when running, together with the display of exaggerated atypic gait pattern were compatible with FMD. Furthermore, the fluctuating course of her condition due to traumatic past events and stressors related to aetiological factors, having consulted neurology clinics for treatment over 4 years without improvement have suggested FMD. During her hospital stay, examination showed improvements in her mobility from time to time with psychiatric treatment, although without enough and maintained recovery. Also, the rigidity and bradykinesia symptoms could not be explained only by FMD. Therefore, the need was seen for detailed neurological tests.

DaTscan, recommended by the neurology unit for differential diagnosis during the treatment process yielded results consistent with PD that helped the patient significantly in improving her clinical symptoms after levodopa treatment. The patient, thought to have FMD and comorbid PD was followed up for both with a planned treatment protocol. In the literature, there are not any cross-sectional epidemiological studies on functional PD (fPD). However, there have been many reported case series indicating high incidences of fPD and PD comorbidity (Parees et al. 2013, Umeh et al. 2013, van der Hoeven et al. 2015). A series of 11 patients presenting with both fPD and PD suggested common neurobiological mechanisms between these two disorders (Parees et al. 2013). Polara et al. (2018) reported a prevalence of 0.64% for fPD cases with PD comorbidity in approximately half of them. Our patient's case was similar to these.

The first step in the investigation of FMD cases is differential diagnosis to assess the presence of a neurological comorbidity. When planning the treatment, the triggering, maintaining, and predisposing factors should be evaluated; and a biopsychosocial model should be formed by taking the patient's mental status, position in family and society, and current life problems into consideration (Başar 2015). The investigation by Spect brain DaTscan was reported to discriminate between fPD and PD (Kägi et al. 2010). The technique was shown to have 98% sensitivity and 67% specificity for PD (de la Fuente-Fernández 2012). DaTscan imaging was helpful in the atypical clinical case of out patient whose differential diagnosis was delayed by 4 years, mainly on grounds of consulting multiple healthcare centers and different physicians without the tenacity of complying with the recommended treatments and follow-up arrangements. The difficulty of diagnosing FMD is associated with the displeasure of the patients in being given a psychiatric diagnosis and the difficulties of inter-clinic communications. The indecisiveness of physicians urge the patients to seek other physicians and further testing. Therefore, it is critical to know how to make a diagnosis, when to continue with further testing, and to come to a definite conclusion (Hallet 2016).

After diagnosing FMD, we emphasized the possibility of a comorbid neurological pathology in our patient on the basis of the common neurobiological factors with PD and the known frequency of comorbidity between the two disorders. The use of imaging and electrophysiological techniques for investigating comorbid neurological disorders after diagnosing

FMD should be remembered. The lack of a common investigative method causes patients to vacillate between neurology and psychiatry clinics as can be seen in the case of our patient which was followed up for 3 years in neurology clinics before referral to psychiatry clinics throughout 4th year. Her condition had worsened by limitation of mobility, impairment of social functionality, and failing to keep up with her daily chores, which, compounded with the loss of hope for a cure, resulted in depression with suicidal ideation and necessitated hospitalisation. Her follow-up on both inpatient and outpatient basis was conducted jointly by the psychiatry and neurology units, facilitating the evaluation of the fluctuations in her FMD symptoms and the effects of pharmacotherapy on her mobility complaints. The case of our patient confirms the importance of a multidiscipilinary approach in dealing with FMD cases (Barbey ve Aybek 2017).

FMD is common and diagnosed with positive evidence. Psychological factors alone are not enough to explain the aetiology of FMD although being critical risk and/or maintaining factors. The approach should be multidisciplinary. Future improvement of the technological equipment of healthcare centers with plans of treating inpatients and outpatients is essential for prioritising the cases of these complex cases.

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