CASE REPORT

Functional movement disorder: a long journey to diagnosis

Sneha Bharadwaj,[1] Melissa Lee,[2] Andrew Campbell Moffat[3]

[1]Department of Geriatric Medicine, Fremantle Hospital, Perth, Western Australia, Australia
[2]Fiona Stanley Hospital, Perth, Western Australia, Australia
[3]Department of Neurology, Fiona Stanley Hospital, Perth, Western Australia, Australia

Correspondence to
Dr Sneha Bharadwaj, sneha.bharadwaj@health.wa.gov.au

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SUMMARY

A 61-year-old man presented to a country clinic with involuntary orofacial movements and progressive cognitive decline, causing significant disability and psychosocial distress. Review of records uncovered a 7-year history of presentations to several specialties, including memory clinics, neurology, internal medicine and emergency departments, with varied symptoms, extensive complex work up and inconclusive diagnosis. Comprehensive review at our hospital highlighted inconsistent neurological signs, fluctuating cognition and psychosocial stressors, which preceded symptom onset, leading to the diagnosis of a functional movement disorder (FMD), which subsequently improved with relaxation therapy, cognitive–behavioural therapy and physiotherapy. We illustrate a variety of somatic symptoms, diagnostic clues and management outcomes for FMDs, and the importance of diagnostic criteria to minimise costly, time-consuming and ultimately unnecessary tests of exclusion.

BACKGROUND

Functional movement disorders (FMDs) are not uncommon; however, lack of clinical experience and access to movement disorder clinics result in prolonged delays in diagnosis, with important implications for the patient and the healthcare system. Such clinical scenarios can be avoided by increasing awareness of a standardised criteria-based approach to diagnosis, and awareness of management options for symptomatic relief.

CASE PRESENTATION

A 61-year-old man presented to a busy country Geriatric clinic with 1-year history of worsening involuntary movements of the face, neck and upper torso associated with mild hemifacial sensory deficits, altered gait, increasing forgetfulness, episodic amnesia and fluctuating attention; this caused disability at work and an inability to provide care to his disabled wife.

On assessment in the country clinic, the patient had frequent bilateral blepharospasm with grimacing on protrusion of his jaw and lower lip, predominantly to the left side, accompanied by painful spasms of platysma and muscles of the upper torso (figure 1A, B), but distractibility was not tested in the clinic setting. There was mild left hemifacial hypoesthesia to pin prick, and slight reduction of power in the left arm and leg (4/5), with an equivocal left plantar reflex. Subtle hemiparkinsonism was noted, with mild increase in tone and cog wheeling at the left wrist, reduced dexterity of the left hand, hesitancy in rising from the chair, slow gait velocity, reduction in left arm swing and episodic shuffling. Speech was variably dysfluent with word finding difficulty. The patient's Montreal Cognitive Assessment (MoCA) score was 13/30 (June 2014) with deficits in all domains. His medical history included previous stroke-like presentation with transient dysphasia and left hemiparesis, chronic headaches, depression and insomnia. He had onset of recent episodic amnesia, confusion and abnormal behaviour, resulting in suspension of his driving license. He was a current heavy smoker with a history of an abusive traumatic childhood in an orphanage.

INVESTIGATIONS

Prior review by a general physician and a neurologist had eventuated extensive investigations.

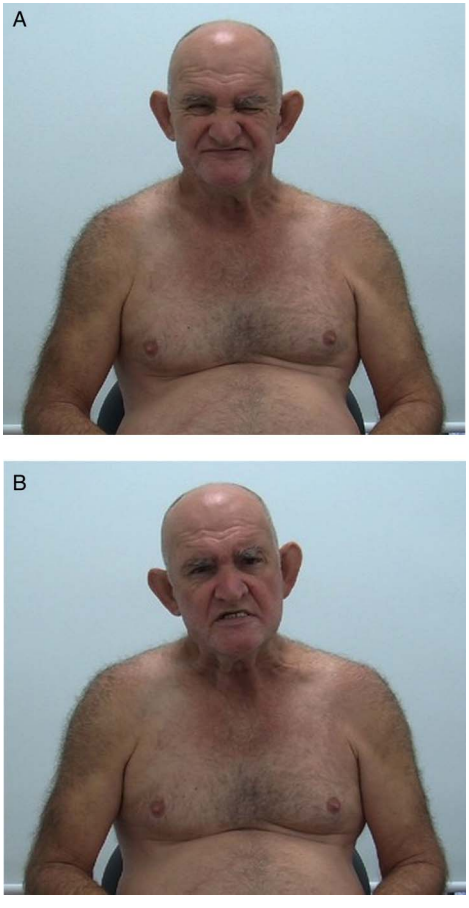

Figure 1 (A) Bilateral blepharospasm and grimacing. (B) Protrusion of lower lip to one side, ipsilateral jaw deviation, and contraction of platysma and sternomastoid muscles.

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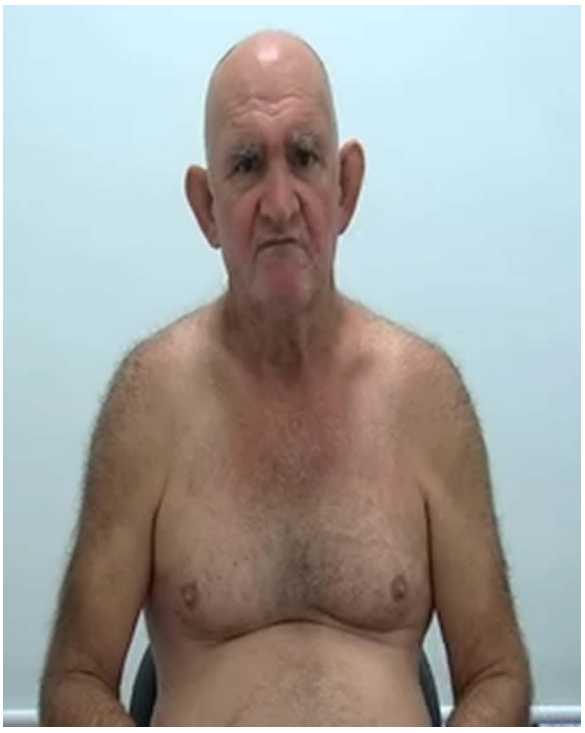

Video 1 Frequent orofacial movements with grimacing, bilateral blepharospasm, spasms of multiple facial muscles, platysma, sternomastoids and muscles of the upper chest. These transient spasms alternate sides and fluctuate in severity and appearance.

Available routine blood tests were normal. Vasculitis and paraneoplastic (glutamic acid decarboxylase 65 and Purkinje cell antibodies) screens were normal, as were tumour markers. Whipple's and HIV serology were negative. EEG showed no abnormal activity. Brain MRI and fluorodeoxyglucose positron emission tomography were unremarkable. Finally, cerebrospinal fluid was negative for protein 14-3-3 and syphilis.

DIFFERENTIAL DIAGNOSIS

The investigations had covered a wide spectrum of differential diagnoses to exclude infective aetiology, paraneoplastic phenomenon, vasculitis, absence seizures or epilepsy, prion pathology, an extrapyramidal disorder or a dementia syndrome; but no conclusive evidence was found to support an organic diagnosis.

OUTCOME AND FOLLOW-UP

On assessment, the varied symptom complex, presentations to other specialties, interval spontaneous remissions, soft neurology, presence of a comorbid mood disorder and disproportionate disability, clearly hinted towards a FMD. Thus, admission to a multidisciplinary geriatric ward was facilitated. During hospital admission, neuropsychological assessment showed flat mood, psychomotor slowing and deficits in visuospatial skills, attention and delayed recall, with inadequate effort during participation. The patient scored 74/105 on the Cambridge Examination for Mental Disorders of the Elderly (CAMCOG), suggestive of a depressive disorder. His MoCA score improved spontaneously to 26/30 (August 2014). Physical examination was remarkably different from that noted in the country clinic 1 month prior. He denied urge preceding orofacial tics, dystonia and myoclonic jerks (video 1), which were noted to improve on distraction, with reduced disability in a supportive environment, providing evidence towards a diagnosis of facial FMD. Hoover's sign could not be demonstrated due to spontaneous resolution of prior neurology. Hemiparkinsonism, noted in the country clinic, had also resolved on admission, negating the need for further investigation with DaT or MIBG scan.

Detailed inquiry identified triggers preceding symptom onset, including severe social and financial stressors leading to declaration of bankruptcy. Previous records from the patient's primary healthcare provider uncovered presentations to varied specialties over 7 years (figure 2), with fluctuating cognitive impairment and 'episodic myoclonic jerks' of the face and upper body. Reassessment by a movement disorder neurologist and neuropsychiatrist was sought, confirming the diagnosis of a FMD consistent with a conversion phenomenon, fulfilling the criteria of the Diagnostic and Statistical Manual of Mental Disorders fifth edition (DSM-V) for conversion disorder. The patient was prescribed relaxation techniques, cognitive–behavioural therapy (CBT) and physiotherapy, which resulted in complete recovery of cognition and significant resolution of the facial FMD over 3 months. He remains well and asymptomatic 1 year following discharge, with a plan for six-monthly ongoing follow-up. He has resumed work and regained his driving license.

DISCUSSION

FMDs are increasingly recognised in the geriatric population. Symptoms vary in severity and characteristics, and may include tremor, dystonia, myoclonus, non-epileptic seizures with short periods of amnesia, gait disorder and Parkinsonism.[1] Diagnosis is challenging and delayed, often being generated by the process of exclusion rather than primary recognition of the disorder.[2 3] Symptom onset can be abrupt, often preceded by triggers, such as physical events, illness or psychological stressors.[4] Clinical characteristics include varied presentations with inconsistency of symptoms, incongruency of signs, spontaneous remissions, fluctuating severity, inadequate effort and symptom exaggeration on

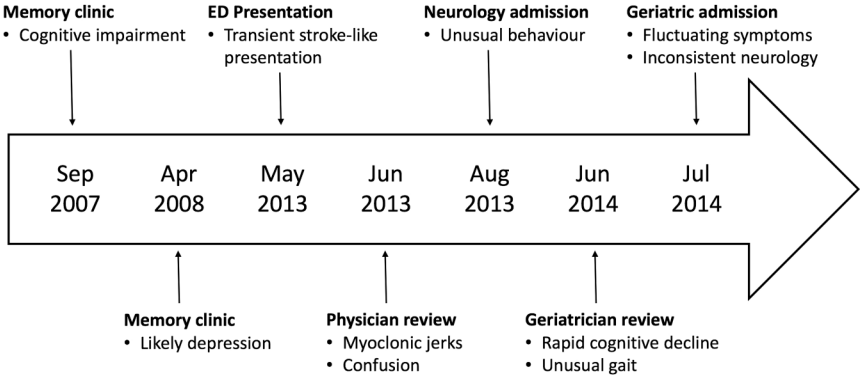

Figure 2 Timeline: presentations and admissions to different specialties, with symptom variability over 7 years (ED, emergency department).

testing with variability over time.[2 4 5] Comorbid depression and psychiatric disorders are common with depressive features and cognitive symptoms causing illness behaviour.[4 6]

Criteria exist to aid clinicians in the diagnosis of FMDs as the primary approach, rather than one of exclusion. The DSM-V criteria for 'functional neurological symptom disorder' neatly integrate the positive features of FMDs,[5 7 8] and no longer require exclusion of malingering and coexistence of psychosocial stressors (prerequisites in DSM-IV for 'conversion disorder').[9] The presence of psychosocial stressors may highlight the possibility of a FMD; however, these should not be relied on for diagnosis, as many patients with structural neurological disease also have psychiatric comorbidities, while several with FMDs may be free of these comorbidities.[10]

Treatment is challenging, with little evidence to guide approach. Providing suitable diagnostic explanation, along with reassurance towards absence of an organic cause, results in overall reduction in hospital attendance and healthcare costs.[9] A multidisciplinary approach is usually required, with emphasis on physical therapy aimed at 'unlearning' abnormal movements.[9 11] FMD-specific physiotherapy is, therefore, useful;[11] and CBT has shown benefit in the treatment of somatoform disorders.[9] Antidepressants may be beneficial and antipsychotics can be considered.[9] Hypnosis, transcutaneous electrical neurostimulation and transcranial magnetic stimulation have been tried in difficult cases, but the evidence for these treatments is unclear.[9]

Prognosis varies with symptom type, but is overall unfavourable, with high residual physical disability and psychological comorbidity. Positive predictors of response to treatment include short symptom duration, early diagnosis and high satisfaction with therapy.[12] Negative predictors are delay in diagnosis and comorbid personality disorder.[12] The involvement of an experienced clinician and a multidisciplinary setting are essential in achieving successful treatment outcome and allowing patients to resume healthy development.[13]

Learning points

- A high index of suspicion is essential in cases with multiple somatic symptoms.
- Inconsistency of signs, fluctuating severity, inadequate effort and symptom exaggeration on testing are diagnostic clues for functional movement disorder (FMD).
- Extensive investigation for a seemingly complex presentation is unnecessary.
- Referral to a movement disorder clinic early on avoids diagnostic delay of FMDs.
- Therapeutic success varies with speed of diagnosis and perceptions of the patient.

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