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Case Reports

Psychogenic Movement Disorders and Dopamine Transporter Scans: Still a Clinical Diagnosis?



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Introduction

Psychogenic movement disorders (PMDs) are managed ideally via a multidisciplinary approach involving primary care providers, specialists in neurology, psychosomatic medicine, physical therapy, and neuropsychology, as well as by collaborating with the patient, family, and acquaintances.¹ Obstacles to efficient therapy include a lack of resources or expertise, differences in the confidence of a PMD diagnosis between neurologists and psychiatrists, and the stigma and patient's response to a PMD diagnosis.

The controversy is ongoing about just how “psychogenic” a PMD is, leading to a debate on the best practices for diagnosis and treatment.² Functional neuroimaging studies show abnormal patterns of brain activation involved in voluntary motor control in patients with functional paralysis or tremor.³ Various psychoanalytic explanations of somatization have been published, such as Winnicott's⁴ defensive dissociation, or a more general view of the mind-body relationship, such as Meissner's.⁵ Given the multiplicity of theoretical models of functional neurologic symptoms and the frequent co-occurrence of psychogenic and neurologic disorders,⁶ ongoing longitudinal assessment of patients with PMD by both neurologists and psychiatrists is crucial for appropriate management.

We demonstrate the importance of ongoing reassessment of PMD through the case of a patient with fixed dystonia, asthma, and noncardiac chest pain who was eventually found to have dementia with Lewy bodies (DLB).

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Mr. A, a 66-year-old Hispanic male patient with a history of anxiety and depression was referred to our movement disorders clinic in May 2012 for neck dystonia and gait disorder evaluation. He had been in good health until the early 2000s when he experienced several health problems, including insomnia, asthma, acute noncardiac chest pain, chronic foot and toe pain, and other vague, chronic pains. His symptoms persisted despite medical treatment by various clinicians. In 2008, his condition became further complicated by symptoms of severe depression and anxiety. Both he and his family had denied cognitive problems. No primary medical diagnosis had ever been established.

His family believed that his situation was owing to the lack of a correct diagnosis, the side effects of medications, and depression. Furthermore, his depression and anxiety were resistant to psychiatric

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treatment as antidepressants and antipsychotics worsened his condition. Often, attempts by his family to bring him into the clinic for behavioral health evaluations provoked asthma attacks, and 1 attempt in 2008 led him to jump from a moving vehicle. During his hospitalization for treatment of that event, a 40-lb weight loss from anorexia was reported.

His medical history revealed a 3-month period of symptom remission, with return to work while on mirtazapine treatment. Unfortunately, he relapsed shortly thereafter. Over the next 2 years (2010–2012), he had a gradual onset of neurologic symptoms with generalized rigidity, gait problems, and slowness. He also had periods of intermittent remission and exacerbation during this time. His family reported that he had experienced more than 10 remissions in a 3-year period. Sudden onset of lateral neck tilting appeared in 2012. Carbidopa-levodopa and quetiapine were started for suspected parkinsonism but caused changes in his personality, hallucinations, agitation, and violence toward his spouse, prompting treatment discontinuation. Risperidone was tried briefly and discontinued because of the recurrence of agitation. He had no other exposure to dopamine-blocking agents that could account for his symptoms. All symptoms except the neck tilt improved after these medications were stopped. He then underwent a dopamine transporter (DaT) scan to characterize his movement disorder, and the result was negative.

When he first presented (in 2012) to our movement disorders clinic, his subjective emphasis was on his neck whereas his family's emphasis was on his depressed mood. He had already visited several neurology centers before presenting to our clinic; botulinum toxin injections in his neck had been tried without any benefit. His medical history also included depression, asthma, gastritis, inguinal hernia repair, and tonsillectomy. His medications included budesonide inhalation suspension, flunisolide nasal solution, and alprazolam (0.5-mg half tablet 3 times daily as needed). His family history was notable in that his mother had an unspecified psychiatric disorder and his brother had an obsessive-compulsive disorder.

During his initial examination in our movement disorders clinic, he was awake, alert, and fully oriented but with marked mutism and lack of engagement in the history taking and examination. This behavior was consistent across several examiners. His examination also revealed weak and variable effort, slowness, passive motion resistance without true rigidity, and variability in

movement velocities over time. There was no true bradykinesia. His neck was fixed in a left laterocollis posture, with no hypertrophy of the left sternocleidomastoid, scalene, levator scapulae, splenius, or trapezius muscles. There was no volitional effort to bring his head back to center. Other examination results were within normal limits. The laboratory assessment results for metabolic causes were negative. We ruled out inflammatory, infectious, autoimmune, endocrinological, electrolyte, and vitamin disorders. Brain and cervical spine MRI scan results were normal.

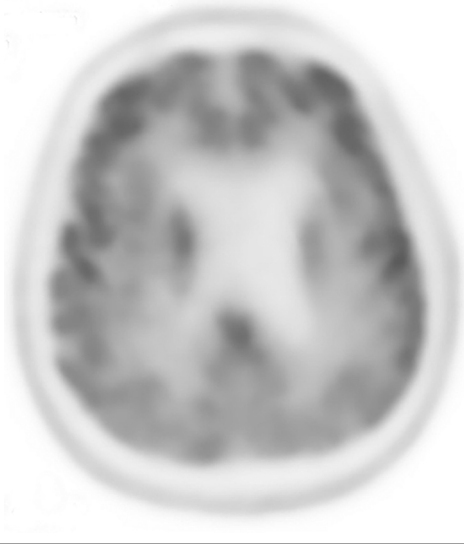
A neuropsychological evaluation in August 2012 revealed performance below expectation on tests of verbal fluency, visuospatial construction, memory, and attention but with inconsistent responses when presented with digit strings of similar length, with a suspected suboptimal effort. The Personality Assessment Inventory disclosed defensiveness and reluctance to admit minor faults and elevation in the somatic concern scale. An electroencephalogram demonstrated a normal posterior dominant alpha rhythm without slowing, lateralization, or epileptiform abnormalities. Treatment focused on his mood, although several psychiatric evaluations were unsuccessful. The diagnostic workup focused on excluding the neurologic causes of his clinical presentation.

A second electroencephalogram 3 years later showed generalized low-voltage slow activity intermixed with symmetric theta and delta waves. During a subspecialized psychiatric evaluation for psychosomatic disorders in May 2015, his speech was notable for short under-elaborated sentences, intermittent mutism, and episodes of verbal perseveration. The Hoffman and grasp reflexes were present bilaterally. On occasion, he demonstrated visual hallucinations and sporadic whole-body jerks. Because of neurologic deficit progression, an ^{18}F -fluorodeoxyglucose-positron-emission tomography (PET) brain scan was performed; it showed occipital lobe hypometabolism consistent with DLB (Figure 1).

Discussion

He was diagnosed with PMD because of symptoms inconsistent with the organic disease model, recurring remissions and exacerbations, psychiatric comorbidity, and negative test results for organic disease, including a negative DaT scan result. In addition, acute fixed dystonias are characteristic of PMDs.^{7,8} Making an

FIGURE 1. ^{18}F -fluorodeoxy glucose-positron-emission tomography showing hypometabolism in the occipital lobe. The darker color density in the frontal lobe indicates normal glucose metabolism, and in contrast, the occipital lobe is lighter, indicating hypometabolism. Used with permission from Barrow Neurological Institute, Phoenix, Arizona.



inclusive diagnosis of PMD is criticized by some, including Stone and Edwards, as an “inappropriately narrow formulation,”² and the correctness of this approach is still debated. Physicians could rush to a presumed PMD diagnosis when there is not enough information to achieve a correct organic diagnosis at a determined time in the disease course. Schrag *et al.*⁷ concluded that fixed dystonias are almost all psychogenic in nature, whereas Pringsheim and Lang recommended caution in diagnosing psychogenic dystonia, given the historical bias until the 1970s that all dystonias were psychogenic.⁹ Among patients with dystonia, the prevalence of PMD ranges from 20–53%. Neurologists specializing in movement disorders use the inclusive criteria proposed by Williams *et al.* to diagnose psychogenic dystonia.¹⁰ These criteria include inconsistency with classic dystonia symptoms and the presence of other psychogenic neurologic signs or symptoms and psychogenic somatic symptoms. Mr. A can be diagnosed with clinically established PMD according to the Williams and Fahn criteria.¹⁰ Moreover, he had developed symptoms over the course of almost a decade before we could conclude that his picture was consistent with DLB.

Our case demonstrates how difficult it may be to diagnose a neurodegenerative condition such as DLB

in its early stages, especially when the patient does not present with the core criteria of fluctuating cognitive impairment, recurrent visual hallucinations, and parkinsonism, and when advanced workups return with negative results. The high baseline intelligence of Mr. A may have allowed him to camouflage his dementia during the early stage of the disease, which further delayed his diagnosis. The coexistence of both disorders is also possible.

Approach to Possible PMD

Key elements in a patient’s history, physical examination, and therapeutic response may prompt consideration of PMD in the differential diagnosis. Patients who typically present with PMD have histories that are inconsistent with organic causes of symptoms. Miyasaki *et al.*¹¹ suggested that after ruling out conditions such as inborn metabolic errors and post-lesion effects, it is the patient history that should alert the clinician to evaluate PMD. Clinicians should consider abrupt onset, static course, spontaneous remissions, obvious psychiatric disturbance, multiple somatizations, presence of secondary financial gain, recent precipitating events, and employment in a health profession as factors indicative of a potential PMD diagnosis.¹¹ This last point is noteworthy because a patient may possess advanced medical knowledge regarding the disease presentation. Caretakers of patients may also have medical knowledge and internalize cardinal features of the disorder. Spontaneous remission is another principal element suggestive of psychogenicity, as very few organic causes demonstrate drastic reductions in physical findings over short periods of time.

After a review of the patient’s history, a physical examination should be done. Significant variations in tremor frequency and rhythmicity were found in 14 patients with psychogenic parkinsonism.⁹ Various techniques can delineate a neurologic tremor from a psychogenic cause. Psychogenic tremors tend to abate if the patient is distracted while performing an action (finger to nose). In organic illness, distracting the patient increases the involuntary movement, although there are exceptions (tics and tardive akathisia) to this rule. Another useful approach is having the patient perform a series of taps at varying frequencies while observing for entrainment, where the “affected” limb takes on the frequency manifested in the unaffected

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limb. Lastly, a patient's response to therapeutic interventions should be monitored.

If the patient's presentation suggests a parkinsonian movement disorder and has elements supporting both organic and psychogenic causes, then a DaT scan may be helpful in guiding appropriate interventions by revealing whether or not the patient is experiencing actual nigrostriatal degeneration. Figure 2 represents our approach algorithm for patients suspected of PMD or parkinsonism.

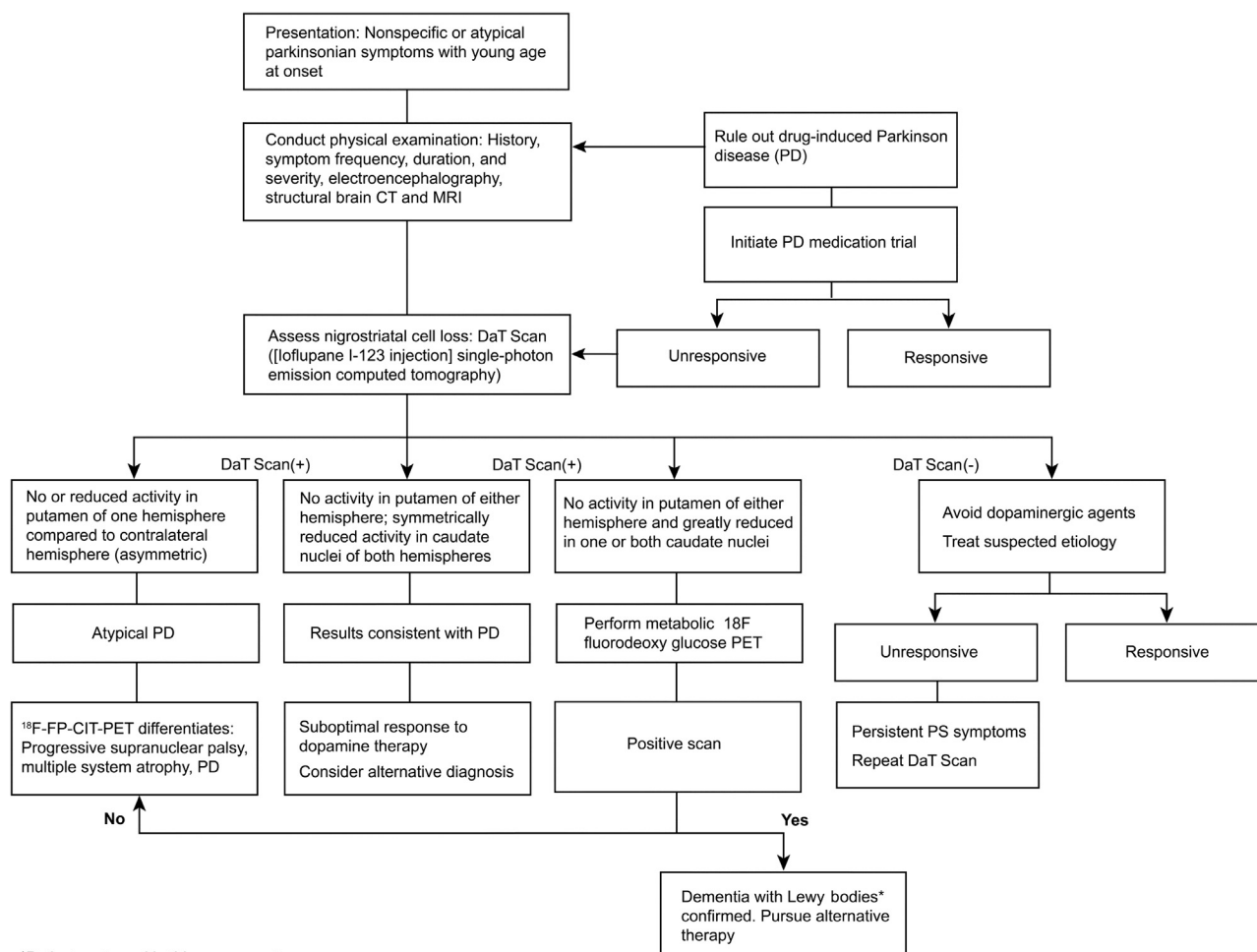
DaT Scans in Movement Disorders

DaT scans are promising in differentiating the dopaminergic nigrostriatal neuron loss of Parkinson

disease (PD) from conditions that mimic PD, but they have intact dopaminergic nigrostriatal regions, such as essential tremor, drug-induced or vascular parkinsonism, and psychogenic parkinsonism.

A DaT scan interpretation requires visual assessment by a trained nuclear medicine radiologist and is considered a highly reliable diagnostic test.¹² A normal DaT scan result should prompt the provider to discontinue any inappropriate medications (e.g., levodopa-carbidopa) and pursue alternate diagnostic tests and therapies to address the patient's underlying condition. However, drug interactions must be taken into account for patients being considered for a DaT as several drugs may interfere with imaging.¹³

FIGURE 2. Diagnostic algorithm for patients with suspected PMD. PS = parkinsonian syndrome; PD = Parkinson disease; ¹⁸F-FP CIT PET = ¹⁸F-fluorodeoxy glucose-positron-emission tomography; PSP = progressive supranuclear palsy; MSA = multiple system atrophy.



A small proportion of patients with idiopathic PD (iPD) may not have presynaptic nigrostriatal degeneration and may have a degree of nigrostriatal degeneration not detectable with SPECT.¹² Although rare, this should be kept in mind when considering rescanning a patient who maintains persistent PD symptoms.

In an open-label, single-dose, prospective, clinical trial of patients with clinically uncertain parkinsonian syndrome, DaT scanning had a sensitivity of 97% and specificity of 100% for differentiating PD from essential tremor.¹⁴ The reasons for clinically uncertain PD included manifesting only 1 of the 3 cardinal signs of parkinsonism, 2 signs without bradykinesia, poor response to L-dopa, lack of disease progression, and atypical signs.¹ Patients who underwent DaT scanning demonstrated a high degree of agreement between the initial scan and the clinical diagnosis 2 years later on follow up.¹⁵ In a study, DaT scanning increased clinicians' confidence in the diagnosis and ultimately led to a change in the clinical management of 72% of patients who had positive scan results.¹⁴

Imaging Studies and DLB

DLB is the second most common cause of dementia after Alzheimer disease. An DLB diagnosis is based on a clinical assessment and is definitively confirmed by autopsy studies. McKeith *et al.*¹⁶ proposed diagnostic criteria for DLB, including the presence of 1 central criterion (dementia) and 2 core criteria (parkinsonian symptoms, persistent well-formed visual hallucinations, or fluctuations in cognition). The core clinical features of DLB reported in the consensus criteria have a specificity of 80% and a sensitivity of 50%. Cognitive decline is not noticeable in the beginning stages of DLB. Thus, it is difficult to make an inclusive diagnosis of DLB using this criterion; suggestive and supportive criteria can be helpful.

Tateno *et al.*¹⁷ identified 3 features that, if present, aid in the diagnosis of possible DLB or disease suggestive of DLB. These features include rapid eye movement sleep behavior disorder, severe neuroleptic sensitivity, and low DaT uptake in the striatum on SPECT or PET. Of these, Mr. A manifested only neuroleptic sensitivity. Significant correlations have been found between decreased striatal DaT levels and visual hallucinations.¹⁸

Our patient also had occipital lobe hypometabolism on PET ([Figure 1](#)), a feature noted in the 2005

DLB consensus guidelines.¹⁶ Chiba *et al.*¹⁹ reported that occipital hypometabolism backs a diagnosis of DLB and perhaps represents the effect of the pathophysiological process of DLB on rapid eye movement sleep behavior disorder. Hypometabolism in the occipital association cortex and primary visual cortex is also reported to have 90% sensitivity and 80% specificity for DLB.²⁰

DaT Scans and DLB

Patients with clinical symptoms highly suggestive of DLB are more likely to have an abnormal DaT scan result than those with possible DLB, although a preexisting high index of suspicion of DLB is usually present before DaT scans are pursued. The clinical criteria and accuracy of diagnosing DLB vary depending on the stage of the illness, which is a primary reason for delaying the referral for an imaging study. Mr. A's DaT scan result was normal at the beginning of his presentation; it would be interesting to know if a subsequent DaT scan result would have been abnormal.

DaT scanning is reported to have an 87% sensitivity in differentiating DLB vs no DLB, with a specificity of 94%.²¹ Others compared imaging results with neuropathological findings in 22 participants and found that an abnormal DaT scan result was 78% sensitive and 90% specific for probable cases of DLB; no participant with a normal DaT scan result had DLB at autopsy.²²

DaT scans may provide false-positive diagnoses of DLB if there is a preexisting vascular lesion in the corpus striatum. DaT scans correlated only with motor dysfunction and, to a much lesser degree, with executive cognitive dysfunction and not with psychiatric or overall cognition.²³

DaT Scans and Psychogenic Movement Disorders

Given the high sensitivity and specificity of DaT scans in PD evaluations, clinicians may use this test to establish the absence of nigrostriatal degeneration in patients with suspected psychogenic parkinsonism, once drug-induced and vascular parkinsonism have been eliminated as the etiology. DaT scans of 5 patients diagnosed with psychogenic parkinsonism, 5 with confirmed iPD, and 5 with healthy controls showed that 2 of 5 patients with psychogenic parkinsonism demonstrated abnormal findings resembling

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those of iPD.²⁴ In 2 of 3 patients with suspected psychogenic parkinsonism who underwent DaT scanning, severe dopamine transporter abnormalities were observed, despite there being scarce clinical evidence of a neurodegenerative etiology.²⁵ In a study of 9 patients with psychogenic parkinsonism monitored for 2 years after having a DaT scan,²⁶ 5 patients whose evaluations clinically qualified them as having established psychogenic parkinsonism had normal DaT scan results. Of the 4 patients with probable psychogenic parkinsonism 3 had normal DaT scan results, and 1 with abnormal findings was eventually diagnosed with iPD. These small studies suggest that DaT scanning is useful to ensure an organic dopaminergic deficit is not missed when psychogenicity is suspected. Although DLB is a diagnostic possibility to explain a patient's abnormal movements, as our case demonstrates, there is a potentially higher risk of false-negative DaT scan results in these patients.

Conclusions

PMD should be diagnosed with caution, especially when DLB (or cognitive compromise) is a

consideration or when the patient presents early in the disease course; the presence of depression in older adults should suggest a more aggressive inclusive diagnosis for neurodegenerative conditions. New imaging modalities such as DaT scans are supportive of certain diagnoses, such as DLB or iPD, but their use and interpretation must always occur in the setting of a multidisciplinary workup in which longitudinal (serial) clinical examinations are the mainstay of diagnosis and treatment.

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