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“Silly walks” in Parkinson’s disease: unusual presentation of dopaminergic-induced dyskinesias

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It is widely recognized that dopaminergic-induced dyskinesias can interfere with gait in advanced Parkinson’s disease (PD).¹ However, levodopa-induced dyskinesias can sometimes take bizarre forms and may easily be mistaken for psychogenic movement disorders. Here, we present four PD patients with strikingly similar bizarre gaits related to the effects of levodopa.

Patient 1 developed clumsiness of the left leg and hand at age 44. He was diagnosed with PD and levodopa treatment was initiated. Six years later, a psychogenic cause was suspected because of bizarre leg movements interfering with stability and gait. We discovered that these dyskinesias mainly occurred at the beginning and end of each levodopa dose effect (Video Patient 1, Segment 1 and 2). In between these periods, the patient experienced motor improvement (Video Patient 1, Segment 3).

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Patient 2 developed hypokinesia and rigidity in the left arm at age 38. Levodopa was initiated, with a good effect on motor function. Three years later, he complained of a short and unpredictable effect of levodopa, usually with a period of immobility and abnormal leg movements preceding advent of the next dose effect (Video Patient 2, Segment 1). After overnight withdrawal of all medication, the patient showed generalized bradykinesia, plus dystonia of the right foot (Video Patient 2, Segment 2). Thirty five minutes after a levodopa dose, ballistic kicking and stamping appeared in the left leg on walking, along with persistent dystonic posturing of the left arm (Video Patient 2, Segment 3). In his full “on” state, the patient showed only mild choreodystonia not interfering with gait (Video Patient 2, Segment 4).

Patient 3 was diagnosed with PD beginning in his left arm at age 35. He started levodopa at age 36 with a good response. At age 41 he developed motor fluctuations and dyskinesias. During the “on” phase he exhibited a striking, stereotyped, kicking gait (Video Patient 3, Segment 1). When “off” he was slow with marked difficulty walking (Video Patient 3, Segment 2). Bilateral deep brain stimulation of the subthalamic nucleus (DBS STN) improved his parkinsonism and allowed reduction of his medications. He no longer exhibits this gait pattern.

Patient 4 developed PD at the age of 39. He responded well to dopaminergic medication, but after four years he developed wearing off and dopaminergic-induced dyskinesias. In his best “on” phase, the patient shows violent choreatic dyskinesias involving the trunk, arms, and legs (Video Patient 4, Segment 1). In the practically defined “off” state, the patient’s gait is clearly bradykinetic, with small, slow and shuffling steps (Video Patient 4, Segment 2). The patient subsequently received bilateral DBS STN, with substantial improvement of dyskinesias and gait.

There are several common elements to these case presentations. The first is the bizarre, yet fairly stereotyped, gait patterns. When videos of these patients were presented at conferences to experienced movement disorder specialists, the gait was generally thought to be psychogenic and reminiscent of the gaits shown in Monty Python’s classic TV sketch “The Ministry of Silly Walks” (<http://www.youtube.com/watch?v=9ZIBUglE6Hc>). The gait we present here indeed looks bizarre, but it is also remarkably stereotyped across patients seen in four different movement disorders clinics. Recurring characteristics include the stepping with kicks, high knee elevations and ballistic ‘stamping’ of one lower limb, generally on the side that was first affected by PD. In addition, dystonic postures of the contralateral foot and ipsilateral arm were seen in two patients, and dancing movements with hops and pelvis rotations in one case.

A further shared feature was that all patients are men, whose PD began before the age of 45 years. In keeping with known experiences with such young-onset patients,² they all developed early motor fluctuations and dyskinesias, starting only several years after initiation of dopaminergic treatment.

Another important element was the timing of these abnormal movements in relation to the intake and clinical efficacy of dopaminergic medication. The two first patients clearly had a diphasic pattern: dyskinesias developed shortly following a levodopa dose, at the beginning of the therapeutic effect. When the levodopa effect increased further to reach a full “on” state, the dyskinesias largely disappeared, and gait improved considerably consistent with previous descriptions of biphasic levodopa-induced dyskinesias that mainly affect subjects with early-onset PD, and that are characterized by a stereotypic pattern, with a dystonic or ballistic form, and a violent expression.^{2–4} The predominance in the most affected leg corresponds to the observation that dyskinesias start and predominate in the leg contralateral

to where dopaminergic denervation in the dorsal putamen is greatest.⁵ Optimizing dopaminergic therapy or DBS can be helpful in these patients, as was also demonstrated here.

Most importantly, the bizarre gait pattern should not be misdiagnosed as a psychogenic movement disorder; even if voluntary movements are sometimes superimposed as a compensation for the dyskinesias interfering with gait (possibly in Patient 4). Another possibility is embellishment which, although not widely recognized, may occur as a superimposed movement abnormality in patients with otherwise genuine PD.⁶ Showing a similar case (#17), Hayes et al.⁷ appropriately warned about the danger of labeling a bizarre gait as psychogenic just on the basis that it is unlike anything one has seen before.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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