Case report

The Dancing Disabling Tongue: Tongue Tremor as a Presentation for Parkinson's Disease

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Abstract

Even though Parkinson's disease may rarely present with tremors of the mandible, the chin and the head, hardly does it present with disabling tongue tremors. We have only come across two cases in published literature. This is a case of a 63-year-old lady who presented with disabling tongue tremors and other unnoticed PD features who responded spectacularly to levodopa/carbidopa. Clinicians should be aware that PD may present with disabling tongue tremors. However, other PD features must be assessed for as PD remains a clinical diagnosis especially in rural third world. A history of neuroleptic use has to be elicited.

Key words: Dancing disabling tongue, Tongue tremor, Parkinson's disease

Introduction

Parkinson's Disease (PD) is a progressive disorder that affects nearly 1% of people aged 60 years and above. Histologically, it is occasioned by the loss of dopaminergic neurons in the substantia nigra due to Lewy bodies. Nevertheless, these Lewy bodies are not confined to the substantia nigra but occur in other parts of the Central Nervous System (CNS), usually starting with the medulla and progressing to the olfactory bulb, pons, substantia nigra, forebrain, amygdala, medial temporal lobe structures with convexity cortical areas affected last (1).

In addition to the four key features of bradykinesia, tremor, rigidity, and gait disturbance there are other associated symptoms such as mood disorders (depression), sleep disorders and autonomic symptoms. Autonomic symptoms that occur in PD are orthostatic hypotension, constipation, difficulty swallowing, urinary retention, and erectile dysfunction. Unilateral tremor occurring at rest with re-emergence is highly specific for PD. Tremor is usually the first sign of PD and occurs fairly early in the evolution of the disorder (2).

Case report

MN was a 63-year-old lady who was brought to the Family Medicine clinic by her father. She was a childless widow who went back to stay with her parents after her husband's death. She had been reportedly well till four weeks before the visit when she was noted to have tremors of the tongue associated with difficulty speaking and swallowing, especially of hot and solid foods. The tremors started insidiously and had progressively worsened over the four weeks. She came to the clinic because the tongue tremors were worsening and affecting her feeding. She was also drooling, and swallowing became progressively more difficult. She was not known to have any chronic medical illness and was not on any medication before the tongue tremors. She didn't report any limb or facial weaknesses of sudden onset. She had noted that occasionally her left hand would have tremors, but she didn't take these seriously but associated them with the passing on of her mother a year ago. She had been the primary caretaker of her father since. She had noticed that she was slowing down in how she walked and did house chores and had had three falls over the last two months. She attributed these falls and slowing down to age. There were neither hallucinations nor signs of dementia. She intermittently got dizzy on standing up. She also had on and off. She neither had constipation nor urine retention.

The patient had a mask-like facial expression as she walked into the office in a slow-shuffling gait with a mild stoop. She had a soft speech that grew softer to the point she got inaudible the longer she talked. It would not be easy to make out what she

was saying. Her father would step in and explain her situation some more. It was noted that her left hand and tongue had obvious tremors at rest. These would disappear on movement but re-occur sometime after movement cessation. There was no cog-wheel rigidity, but she had difficulties turning around when asked to walk to the door and back. Her blood pressure was slightly elevated at 158/90 mmHg, but no postural hypotension was elicited on repeating the BP when she was standing up. She was not able to write, and thus, micrographia couldn't be elicited. The basic panel of full blood count, urea and electrolytes, random blood sugar and lipid profile were all normal. A brain MRI was requested, but it was not done due to cost constraints. The patient was diagnosed with Parkinson's disease on clinical grounds, and levodopa/carbidopa was initiated with marked improvement of tongue tremors and general mobility. The cost of the drug is another barrier that they will have to surmount as PD is chronic and progressive, the need for the drug is long-term, and there will be a need to up-titrate the dosages (and thus cost) or add other medicines for optimal control of symptoms. She was the first person to be diagnosed with PD in both their immediate and extended family.

Discussion

In 2015, Jaulent and colleagues (3) wrote the first case report of 'de novo PD revealed by a disabling tongue tremor'. They opine that even though Parkinsonian rest tremors can affect the mandible, the chin and the head, they were the first to report it (the PD tremor) involving the tongue in a disabling manner. This patient was not on any neuroleptics; thus, the tremor couldn't be a consequence of drug use, as has been reported in other case reports (4).

The other differential is essential tremor. However, its tongue tremors do not present with disabling symptoms (5). In addition, this patient had other features characteristic of PD, and the tremor was re-emergent, all of which seemed to exclude this differential (6). One other major differential is oromandibular dystonia. However, the patient did not report problems with jaw opening or closing or blepharospasms. Again, as noted above, other features of PD and associated response to levodopa/carbidopa make PD the more likely diagnosis (7).

Parkinsonian tremors respond well to levodopa/carbidopa and dopamine agonists used in PD management. In case of severe tremors, PD patients may have to undergo thalamic deep brain surgery to ameliorate the symptoms. This is the case in seemingly milder forms of PD, like benign tremulous parkinsonism (8).

It is postulated that the pattern of motor symptoms in PD is determined by the severity and distribution of neuronal degeneration in specific areas of the substantia nigra, and that this process is usually faster in regions corresponding to the upper limbs. Delil and colleagues (6) propose that, on occasion, areas related to the tongue may experience neuronal degeneration before the traditional priority areas, i.e. the limbs.

Given that this is a case report, causation cannot be definitely established. The proposed mechanism of occurrence is purely speculative. Additionally, there is risk of recall bias, as both the clinician and the patient are likely to remember certain details more vividly than others. There is also a tendency to construct a coherent narrative around the case to increase the chances of publication. Furthermore, journals may not publish all case reports, which could lead to publication bias.

Conclusions

As clinicians, we must be aware that PD may present with disabling tongue tremors. We must evaluate for other PD features not narrated by the patient, not forgetting antipsychotics as a major cause and essential tremor and oromandibular dystonia as key differentials. PD remains a clinical diagnosis as tests aim to identify certain differential diagnoses.

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