Paraspinal Muscle Asymmetry in Parkinson's Disease

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ABSTRACT

OBJECTIVE: To evaluate the radiographic anatomy of paraspinal asymmetry in PD. **BACKGROUND:** Postural changes are a well known levodopa unresponsive feature of PD. Although forward deviation (flexion) of the spine is most obvious, lateral deviation is also common. This has never been radiographically queried to evaluate for specific muscle involvement and pathology. **METHODS:** We initially identified 15 PD patients from the Baylor College of Medicine Movement Disorders Clinic whom on examination demonstrated paraspinal asymmetry and variably laterally deviated posture. Lumbar and/or thoracic MRIs were performed to evaluate relative paraspinal muscle mass and morphology. **RESULTS:** Nine subjects (8 female) were identified with paraspinal asymmetry on examination and had MRI spine imaging. The age at time of imaging was 74.8±8.8 years, following 7.1±3.1 years since the onset of PD. Relative atrophy was seen diffusely in all paraspinal muscles (psoas, interspinalis, quadratus, multifidus, longissimus, and ileocostalis). The quadratus, multifidus, longissimus, and ileocostalis were the most asymmetric and equally involved. The interspinalis was less asymmetric, whereas the psoas was almost never asymmetric. Fatty infiltrates, consistent with myopathic degeneration, were often seen in the atrophic muscles. In 8/9 cases the side of PD symptom onset demonstrated the greatest atrophy. **CONCLUSION:** Lateral postural deviation appears to result from ipsilateral paraspinal atrophy with fatty infiltration. This occurs in the side first affected by PD. Given this structural abnormality, it is unlikely that symptomatic pharmacological therapy could benefit posture. The relationship between this phenotype and camptocormia in PD is not clear.

INTRODUCTION

Postural changes are a well known levodopa unresponsive feature of PD. Although forward deviation (flexion) of the spine is most recognized, lateral deviation is also common, and often associated with grossly observable and palpable asymmetry in paraspinal bulk. This has never been clinically evaluated or radiographically queried to evaluate for specific muscle involvement and pathology.

METHODS

We identified fifteen PD patients from the Baylor College of Medicine Movement Disorders Clinic who upon examination demonstrated paraspinal asymmetry and variably laterally deviated posture. Patients with thoraco-lumbar flexion of greater than 20º ("camptocormia" or "bent spine") were excluded. We also excluded patients with any surgical intervention or other neurological history that could result in abnormal muscle bulk prior to the imaging. Historical and demographic data were obtained and all underwent the motor examination of the Unified Parkinson's Disease Rating Scale (UPDRS). We also rated dopaminergic treatment responses (0=no response, 1=minimal improvements, 2=moderate improvement, 3=marked improvement but no history of dyskinesia, 4=marked improvement with dyskinesia).

In three, lumbar and/or thoracic MRI imaging had been performed within the past year and in the remainder it was obtained when possible. The paraspinal muscle mass of all major paraspinal muscles was rated as 0-3, maximum score of 18 for the 6 muscles. MRIs were independently read by a neuroradiologist (HH) blinded to clinical features.

RESULTS

Of the 15 identified patients, two females were excluded because they could not have a lumbar MRI secondary to deep brain stimulation, one female was excluded because of a previous lumbar laminectomy, and three (one female) did not demonstrate paraspinal asymmetry on MRI imaging, despite asymmetry on physical examination. In the remaining 9 patients (8 female) the age at time of imaging was 73.1±10.7 years, following 7.0±3.1 years since the diagnosis of PD. [Table 1] All but one patient reported non-radiating low back pain. In 8/9 patients the relatively more atrophied side was the initially affected symptomatic side for their PD symptoms.

MRI findings in the patients are summarized in Table 2 and shown in Figure 1. Relative atrophy was seen in all paraspinal muscles (psoas, interspinalis, quadratus, multifidus, longissimus, and ileocostalis). The quadratus, multifidus, interspinalis, and ileocostalis were the most asymmetric and equally involved. The longissimus was moderately less asymmetric, whereas the psoas was the least asymmetric. In most cases, fatty infiltrates were seen in the relatively more atrophied muscles. The more robust asymmetry was usually seen at the L2-L3 level.

Figure 1. MRI findings in patients.

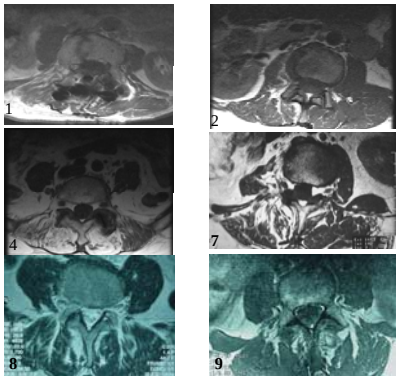

Table 1. Clinical summary.

	Sex	Age	Age of PD Onset	Side of Onset	Initial Sign	Levodopa Response	Total UPDRS Motor	Right Paraspinal Size	Left Paraspinal Size
1	F	83	80	R	Gait	2	29	9	18
2	F	77	68	R	Bradykinesia	3	21	6	13
3	F	62	53	R	Tremor	2	40	12	18
4	F	83	72	R	Tremor	4	40	10	13
5	F	76	67	R	Tremor	3	53	11	12
6	F	58	55	R	Gait	2	30	10	14
7	F	79	69	L	Tremor	4	40	8	18
8	F	78	67	R	Tremor	4	53	14	16
9	M	77	74	L	Tremor	3	33	15	12

Table 2. Summary of paraspinal asymmetry.

	Psoas		Interspinalis		Quadratus		Multifidus		Longissimus		Ileocostalis		TOTAL	
	R	L	R	L	R	L	R	L	R	L	R	L	R	L
1	3	3	1	3	1	3	1	3	1	3	2	3	9	18
2	2	3	1	2	0	2	0	2	1	2	2	2	6	13
3	2	3	2	3	2	3	2	3	2	3	2	3	12	18
4	3	2	1	1	1	1	2	3	2	3	1	3	10	13
5	3	3	1	1	1	1	2	2	2	2	2	3	11	12
6	3	3	2	2	1	2	1	2	1	2	2	3	10	14
7	2	3	1	3	1	3	1	3	2	3	1	3	8	18
8	3	2	1	3	2	3	3	2	3	3	2	3	14	16
9	3	2	1	2	3	2	2	2	3	2	3	2	15	12

CONCLUSIONS

In this preliminary study we assessed the anatomy that results in paraspinal asymmetry seen on examination in some patients with PD. Overall, we found generalized relative atrophy with fatty infiltrations more abundant in the initially symptomatic parkinsonian side. Women were disproportionally represented. We did not attempt to quantify what percentage of PD patients had overt paraspinal asymmetry on examination but identified 15 subjects in approximately 12 weeks. It is more common than camptocormia in our clinic.

To our knowledge this is the first report focusing on paraspinal asymmetry in PD. The fatty infiltrates, seen mostly on the atrophied side, are similar to those reported bilaterally in some cases of camptocormia, which has been associated with PD. (1-7) Radiographically, this pattern is most consistent with myopathic changes, as opposed to neuropathic atrophy, which maintains normal muscle density. (8) The most asymmetric spinal level, L2-L3, is similar to the most atrophied areas seen in general camptocormia (9), perhaps suggesting some intrinsic vulnerability at that level

CONCLUSIONS

It is not clear whether this paraspinal asymmetric atrophy represents the same process as camptocormia, but that only occurs unilaterally and thus does not cause kyphosis, or whether it is completely distinct entity. Compared to series of camptocormia in PD, our patients were more likely to present with tremor, were more likely to initially present with unilateral symptoms, as is typical for PD, were more likely to be female, and had a shorter duration of PD. (1) Therefore it is possible that our series represents the same pathology set upon a different underlying baseline, or is simply a "forme fruste" of overt camptocormia.

The pathophysiology of camptocormia and dropped head syndrome seen in parkinsonism is debated and probably multi-factorial. Camptocormia in parkinsonism has been attributed to both increased muscle flexion force, usually dystonia (1, 4, 7), and decreased extension force, usually attributed to various myopathic processes. (5, 6, 10, 11).

The CNS motor control of paraspinal muscles is unique. Although these muscles are controlled by pyramidal pathways they are also heavily innervated by the reticulospinal and vestibulospinal tracts, which are thought to modulate baseline posture. These tracts are subserved by the pedunculopontine tegmental nucleus, a major outflow from the basal ganglia. However, neither dopaminergic medications nor deep brain stimulation of the sub-thalamic nucleus, markedly improve posture in PD. This is not surprising given the morphological changes in our series and in some with camptocormia in PD.

The natural morphology of paraspinal muscle mass in the general population is not well studied but appears to gradually atrophy with age, as do other muscles. (8, 12) Asymmetry has not been reported in the normal population. Mild fatty infiltrates are increasingly seen as people age but are not distributed in the muscle center as was seen in our population. (9) Furthermore, the correlation between the side of PD onset and atrophied side suggests a culpable pathology rather than chance occurrence. Future studies should establish the prevalence of this within the PD population, elicit clinical and electrophysiological correlates of the sign, query for specific myopathic or neuropathic degeneration, follow these patients over time to see if they develop camptocormia, and specifically evaluate for treatment responses, including steroid therapy.

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