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Title Page

Accuracy of clinical diagnosis in tremulous parkinsonian patients: a blinded video study.

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Abstract: (237 words)

This study examines the clinical accuracy of movement disorder specialists in distinguishing tremor dominant Parkinson's disease (TDPD) from other tremulous movement disorders by the use of standardised patient videos.

Two movement disorder specialists were asked to distinguish TDPD from patients with atypical tremor and dystonic tremor, who had no evidence of presynaptic dopaminergic deficit (subjects without evidence of dopaminergic deficit or SWEDDs) according to ^{123}I -N- ω -fluoro-propyl-2 β -carbomethoxy-3 β -(4-iodophenyl) nortropane ($[^{123}\text{I}]$ FP-CIT) SPECT, by 'blinded' video analysis in 38 patients.

A diagnosis of Parkinsonism was made if the step 1 criteria of the Queen Square Brain Bank criteria for Parkinson's disease were fulfilled. The reviewer diagnosis was compared to the working clinical diagnosis drawn from the medical history, SPECT scan result, long term follow-up and in some cases the known response to dopaminergic medications. This comparison allowed a calculation for false positive and false negative rate of diagnosis of PD.

We found a high false positive (17.4 to 26.1%) and negative rate (6.7 to 20%) for the diagnosis of PD. The diagnostic distinction of TDPD from dystonic tremor was reduced by the presence of dystonic features in treated and untreated PD patients.

Clinical distinction of TDPD from atypical tremor, monosymptomatic rest tremor, and dystonic tremor can be difficult, due to the presence of parkinsonian features in tremulous SWEDD patients. The diagnosis of bradykinesia was particularly challenging. This study highlights the difficulty of differentiation of some cases of SWEDD from PD.

Introduction

Clinico-pathological correlations have improved the diagnostic accuracy of Parkinson's disease (PD); in tertiary referral movement disorder clinics there is close to 100% concordance between the final clinical diagnosis and pathological findings.[1,2] Nonetheless patients with tremor dominant Parkinson's disease (TDPD) continue to be a diagnostic challenge and are under-represented in published post-mortem studies.

In a community based study in Wales,[3] only 53% of patients, treated with anti-parkinson therapy in primary care, met the Queen Square Brain Bank criteria (QSBBC) for the clinical diagnosis of PD when re-examined by an experienced movement disorder specialist. A quarter of the cases had no signs of parkinsonism and half of these were considered to have essential tremor (ET).

It is likely that a significant proportion of the 4-15% of *de novo* patients recruited for recent clinical trials with anti-parkinsonian drugs who have no evidence of nigrostriatal dopamine denervation on functional imaging (subjects without evidence of dopaminergic deficit or SWEDDS) have tremulous syndromes other than PD. This is supported by long term follow up showing minimal clinical deterioration, no response to dopaminergic therapy and in some cases no evidence on repeat functional imaging of dopamine denervation.[4,5]

In this study, the clinical features of patients with tremor and parkinsonism were assessed by two blinded movement disorder specialists (DGG and AJL). The diagnosis arrived at by this process was compared in each case with the working clinical diagnosis (derived in all cases from history and examination by a third movement disorder expert [NB], long term follow up and [¹²³I] the FP-CIT result).

Methods

Participants and On-site Diagnosis

A consecutive group of patients with clinically uncertain Parkinson's disease were prospectively recruited from the neurology clinics in Nottingham and Derby (local ethics approval 07/Q2502/1). Patients were referred from both primary care (75%) and secondary (25%) care. Study inclusion was based on clinical diagnostic uncertainty, including the presence of prominent postural tremor, or focal, multifocal or segmental dystonia. Patients exposed to dopamine receptor antagonists or toxins such as manganese or carbon monoxide were excluded. Cases considered psychogenic, or with uncommon secondary or atypical causes of parkinsonism (e.g. Huntington's disease, fragile X ataxia syndrome) were also excluded.[6-13] Neurological examination was repeated after an average of 3 years of follow up, and structural imaging used selectively to exclude causative cerebral lesions (e.g. supratentorial meningioma, extensive cerebrovascular disease)(MR/CT brain imaging conducted in 15/38 cases).

The patients were collected over a three year period and referred for [¹²³I] FP-CIT scan in accordance with NICE (National Institute for Health and Clinical Excellence) guidelines.[14] All patients gave written informed consent for use of their video material for research and teaching.

All patients were examined by one of the authors (NB) and a final diagnosis of PD, dystonic tremor, atypical tremor, monosymptomatic rest tremor or ET made. This was designated the working clinical diagnosis. All patients with a

working clinical diagnosis of PD were treated with L-dopa and/or dopamine agonist drugs over the three year follow-up period. In addition, a minority of patients who were subsequently re-classified as non-PD, had had a trial of dopaminergic therapy prior to initial referral (9 of 23 cases, 39%).

Blinded Reviewer Diagnosis

Video recording of all patients was performed according to standard criteria for the Unified Parkinson's Disease Rating Scale (UPDRS) part III (motor score) and to demonstrate the features of essential tremor and dystonic tremor according to consensus criteria.[15] At the time the videos were taken, no non-PD patient was on dopaminergic therapy. The minority of PD patients on dopaminergic therapy at the time of the video were excluded from final analysis of false-negative patients with PD, in case treatment masked their clinical features.

Two movement disorder specialists (AJL and DGG) were asked to suggest the likely clinical diagnosis based on video footage. Their diagnoses took into account the QSBB operational criteria for the diagnosis of PD (with the exception of rigidity), the consensus criteria for essential tremor and clinical features suggestive of dystonic tremor.[15][16][17] The term dystonic tremor is used in this paper to embrace both "dystonic tremor" and "tremor associated with dystonia" as defined previously.[15,18]

The abnormalities observed on sequential movement testing were broadly divided into bradykinesia typical of PD (slowness with motor decrement) or

atypical of PD (pure slowness, slowness with delay in initiation of fine finger/hand movements or slowness with reduced amplitude of fine finger/hand movements without decrement). The term flurry or flurries were used to describe the rapid change in tremor amplitude and frequency that is a common feature of dystonic tremor.[17,19,20] Since re-emergence of postural tremor is sometimes considered to be the postural representation of rest tremor in PD, the time to emergence of postural tremor was calculated visually by both reviewers for all 38 cases.[21]

Imaging Analysis

[¹²³I] FP-CIT imaging was carried out according to standard protocols with discontinuation of agents that might interfere with [¹²³I] FP-CIT striatal uptake at least 4 weeks before the scan. Thyroid blocking and single slow intravenous injections of [¹²³I] FP-CIT were administered at activities between 111 and 185 MBq. Images were acquired 3-6 hours after injection using a multi-headed gamma camera.

All [¹²³I] FP-CIT scans were scored in routine clinical service, and then independently by a second nuclear medicine specialist blind to both clinical information and video footage (JP). Visual assessment graded scans as normal or abnormal (scale 0-3).[22]

Statistical Analysis

We ascertained inter-reviewer agreement on video diagnosis using Cohen's kappa coefficient for each pair of blinded reviewer diagnoses per video case.

The blinded reviewer diagnoses based on video were also compared with the working clinical diagnoses. This allowed calculation of a false positive and negative rate with respect to the diagnosis of PD for the two movement disorder experts by standard formulae. The clinical and imaging data were then shared with the blinded experts.

Results

Blinded second read of [¹²³I] FP-CIT SPECT

The second read of all 38 FP-CIT SPECT scans of patients (JP) showed full concordance in abnormal versus normal with the initial read of scans by the local nuclear medicine specialist (JB). Of the fifteen abnormal scans, there was agreement on the grade of abnormality in 8 and disagreement in 7 (table 1). The scale of disagreement was never more than one point. Only two of these 6 cases of disagreement of scale featured in the false negative cases (tables 3b and 4b). Scan reports in all cases showed congruence in the most affected side on the FP-CIT scan and the clinically most affected side.

Working clinical diagnosis

In the case of TDPD patients, the working clinical diagnosis was based on clinical observation for an average of 39.6 months (SD 20.7), as well as review of video recordings (9 were recorded an average of 13.9 months after the FP-CIT scan and in 6 others, an average of 8.3 months before the FP-CIT scan); the L-dopa/dopamine agonist response and in all cases FP-CIT scan evidence of dopaminergic deficit.

Similarly, the working clinical diagnosis for clinically uncertain PD patients was based on a normal FP-CIT scan and clinical observation for an average of 35.9 months (SD 19.3); videos were taken an average of 8.8 months after the scan in 12 cases and in 11 cases an average of 5.1 months before the FP-CIT scan. In addition, a minority of patients had a trial of dopaminergic therapy (9 of 23 cases, 39%).

Blinded reviewers' diagnosis

The blinded baseline diagnosis on video assessment for each of the 38 cases for reviewers 1 and 2 is listed in table 1.

Table 1. Clinical Diagnosis on blinded video assessment for 38 cases for Reviewer 1 and 2 versus FP-CIT result and the working clinical diagnosis.

Case	Blinded Video Diagnosis		FP-CIT Result	Working Clinical diagnosis
	R1	R2		
1	DT	PD	Normal	DT
2	ET	DT	Normal	DT
3	PD	PD	Abnormal (grade 3 JP; grade 2 JB)	TDPD
4	DT	DT	Normal	DT
5	DT	PD	Abnormal (grade 1)	TDPD
6	DT	DT	Normal	DT
7	Monosymptomatic rest tremor	DT	Normal	ET
8	DT	DT	Normal	DT
9	PD	DT	Abnormal (grade 1)	TDPD
10	DT	DT	Normal	DT
11	DT	DT	Normal	ET
12	PD	PD	Abnormal (grade 2 JP; grade 1 JB)	TDPD
13	ET	PD	Normal	DT
14	Monosymptomatic rest tremor	PD	Abnormal	TDPD

			(grade 2 JP; grade 1 JB)	
15	ET	DT	Normal	DT
16	ET	DT	Normal	DT
17	PD	DT	Normal	DT
18	Atypical Tremor	PD	Abnormal (grade 2)	TDPD
19	DT	DT	Normal	DT
20	DT	DT	Normal	ET
21	PD/ Atypical Tremor	PD	Abnormal (grade 2 JP; grade 3 JB)	TDPD
22	DT	DT	Normal	DT
23	PD	PD	Normal	ET
24	DT	PD	Abnormal (grade 2)	TDPD
25	DT	DT	Normal	DT
26	DT	PD	Normal	DT
27	DT	DT	Normal	DT
28	PD	PD	Abnormal (grade 3 JP; grade 2 JB)	TDPD
29	ET	DT	Normal	DT
30	PD	PD	Normal	DT
31	PD	PD	Abnormal (grade 2 JP; grade 1 JB)	TDPD
32	PD/Atypical Tremor	DT	Normal	ET
33	PD	PD	Abnormal (grade 2)	TDPD
34	Atypical tremor	DT	Abnormal (grade 1)	TDPD
35	ET	DT	Abnormal	TDPD

			(grade 1 JP; grade 2 JB)	
36	PD	PD	Abnormal (grade 2)	TDPD
37	ET	PD	Abnormal (grade 2)	TDPD
38	ET	PD	Normal	DT

PD = Parkinson's Disease; DT = Dystonic Tremor; TDPD = Tremor Dominant Parkinson's Disease; ET = Essential Tremor; R1 = Reviewer 1; R2 = Reviewer 2; Grades 0-3 for FP-CIT.[22]; JP and JB refer to nuclear physicians across the two sites.

Comparison of reviewers' diagnoses with the working clinical diagnosis

There were differences in sensitivity and specificity of the blinded diagnosis based on video assessment between the two reviewers (Table 2). Reviewer 2 had a higher sensitivity for detecting PD, with an associated lower specificity, compared to reviewer 1.

Table 2. Diagnosis of PD in 15 patients, based on blinded video review compared to working clinical diagnosis

	Video Diagnosis	
	Reviewer 1	Reviewer 2
False Positive diagnosis of PD	17.4%	26.1%

False Negative diagnosis of PD	46.7%	20.0%
False Negative diagnosis of PD, after excluding 5 patients on dopaminergic therapy	20.0%	6.7%
Sensitivity for PD	53.3%	80.0%
Sensitivity for PD, after excluding 5 patients on dopaminergic therapy	72.3%	93.3%
Specificity for PD	85.2%	79.3%

Tables 3a/b and 4a/b illustrate the clinical features which led the reviewers to a baseline video diagnosis which was incompatible with the FP-CIT SPECT result and the working clinical diagnosis.

Table 3a. False positive diagnosis of PD in 4 cases on blinded video review.

Reviewer 1

False Positives	Blinded video Diagnosis	Clinical features leading to diagnosis				FP-CIT result
		FB	FH	RAS	GI	
Case 17	PD	+	+	-	+	Normal

Case 23	PD	+	-	+	-	Normal
Case 30	PD	+	+	-	-	Normal
Case 32	PD/ Atypical ET	-	+	-	-	Normal

PD = Parkinson's Disease; ET = Essential Tremor; DT= Dystonic Tremor; FB = Fatiguable bradykinesia; FH = Facial hypomimia; RAS = reduced arm swing; GI = Gait impairment; RT = rest tremor; HV = hypophonic voice

Table 3b. False negative diagnosis of PD in 7 cases on blinded video review.

Reviewer 1

False Negatives	Blinded video Diagnosis	Clinical features leading to diagnosis						FP-CIT Result	Drugs at time of video			
		N	P	R	T	F	B			M	F	N
Case 5	DT	-	+	-	-	+	Abnormal (grade 1) Congruent	Drug naive				
Case 14	Monosymptomatic Rest Tremor	+	+	+	-	-	Abnormal (grade 2 JP; grade 1 JB) Congruent	Drug naive				
Case 18	Atypical Tremor	+	+	-	-	-	Abnormal (grade 2) Congruent	6 months of pramipexole (LEDD of 213mg/day) ⁱ				
Case 24	DT	-	-	-	-	+	Abnormal (grade 2) Congruent	On L-dopa for 3 years 400mg/day				
Case 34	Atypical Tremor	-	+	+	-	-	Abnormal (grade 1) Congruent	8 months of L-dopa (300mg/day)				
Case 35	ET	-	+	-	-	-	Abnormal (grade 1 JP; grade 2 JB) Congruent	Drug naive				
Case 37	ET	-	+	-	+	-	Abnormal	2 months of				

						(grade 2) Congruent	L-Dopa 300mg/day
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¹LEDD= Levodopa Equivalent Daily Dose (Pramipexole daily dose x 67)

NPRT = no pill rolling rest tremor; NFB = no fatiguable bradykinesia; MF = myoclonic flurries; NGI = no gait impairment; DF = dystonic features e.g. collis, thumb hyperextension, shoulder elevation; Congruent refers to predominant FP-CIT abnormality contra-lateral to most affected clinical side

Table 4a. False positive diagnoses of PD in 6 cases on blinded video review.

Reviewer 2

False Positives	Blinded video Diagnosis	Clinical features leading to diagnosis						FP-CIT Result
		FB	RT	HV	FH	RAS	GI	
Case 1	PD	+	-	-	-	-	-	Normal
Case 13	PD	-	+	-	-	+	-	Normal
Case 23	PD	+	-	-	-	+	-	Normal
Case 26	PD	+	-	+	-	+	-	Normal
Case 30	PD	+	-	-	-	-	-	Normal
Case 38	PD	+	-	-	-	+	-	Normal

PD = Parkinson's Disease; ET = Essential Tremor; DT= Dystonic Tremor; FB = Fatiguable bradykinesia; FH = Facial hypomimia; RAS = reduced arm swing; GI = Gait impairment; RT = rest tremor; HV = hypophonic voice

Table 4b. False negative diagnosis of PD in 3 cases on blinded video review.

Reviewer 2

False Negatives	Blinded video Diagnosis	Clinical features leading to diagnosis	FP-CIT Result	Drugs at time of video

		NPRT	NFB	MF	NGI	DF		
Case 9	Dystonic Tremor	-	+	-	-	+	Abnormal (grade 1) Congruent	12 months on L-dopa 300mg/day
Case 34	Dystonic tremor	-	+	-	-	-	Abnormal (grade 1) Congruent	8 months on L-dopa 300mg/day
Case 35	Dystonic Tremor	+	+	-	-	-	Abnormal (grade 1 JP; grade 2 JB) Congruent	Drug naive

NPRT = no pill rolling rest tremor; NFB = no fatiguable bradykinesia; MF = myoclonic flurries; NGI = no gait impairment; DF = dystonic features e.g. collis, thumb hyperextension, shoulder elevation; Congruent refers to predominant FP-CIT abnormality contra-lateral to most affected clinical side

Analysing the false positive case errors, the clinical feature common to 8 of the 10 cases was the detection of apparent bradykinesia. Only 2 false positives were common to both reviewers (cases 23, 30).

Analysis of the 10 false negative cases is complicated by the fact that 6 patients were on dopaminergic therapy. Within the drug naïve false negative cases, reviewer 1 recorded slowness but no decrement in amplitude, and thumb hyperextension, in case 5; no bradykinesia, flurries, and non-pill-rolling tremor in case 14; no bradykinesia, absence of re-emergent tremor, and

normal gait in case 35. Reviewer 2's conclusions for case 35 were similar, that bradykinesia was not present and that there was a suggestion of retrocollis.

Considering the false negative cases on dopaminergic therapy, case 34 was the only case falsely diagnosed by both reviewers. Case 34 was felt by reviewer 1 to have flurries of tremor and no bradykinesia and by reviewer 2 to have no bradykinesia.

Bradykinesia

Summarising the findings of reviewer 1, 12 cases were considered to have true bradykinesia. Of these 12 cases, only 8 had presynaptic dopaminergic deficit on FP-CIT SPECT. For reviewer 2, 21 cases were considered to have true bradykinesia, of which only 10 had FP-CIT scans indicating presynaptic dopaminergic deficit. A total of 3 cases with normal FP-CIT scan were rated as having true bradykinesia by both reviewers. True parkinsonian bradykinesia was therefore represented in both patients with normal and abnormal FP-CIT scans.

Slowness of a non-PD type is reported in dystonic tremor.[17] Of the non-PD bradykinesia types in this series, only pure slowness without fatiguing or reduced amplitude of movement was discriminating for patients with normal FP-CIT scan.

Re-emergent tremor

The average latency to postural tremor across the two blinded reviewers for the 23 SWEDD patients was 0.82s (median 0s, interquartile range 0-2s) and for the 15 TDPD cases the latency was 0.79s (median 0s, interquartile range 0-0.5s)($p = 0.69$ Mann-Whitney U Test). There was no significant difference in latency between SWEDD and TDPD cases for re-emergent tremor.

Inter-blinded reviewer agreement on video diagnosis

The inter-blinded reviewer agreement on video diagnosis (PD or non-PD) was low (Kappa coefficient = 0.24 95%CI = 0, 0.48).

Discussion

The patients with suspected Parkinsonism used in this study were divided into those with normal (scans without evidence of dopaminergic deficit; SWEDDs) or abnormal presynaptic dopamine activity. Although supra-nigral forms of Parkinsonism can have a normal FP-CIT scan, few of these have rest tremor as the predominant feature and the differential diagnosis for the SWEDDs in this study lay mainly between dystonic tremor, ET and atypical tremor.

Of the other diagnoses proffered by the reviewers, monosymptomatic rest tremor is considered by many authorities as a *forme fruste* of PD and may have abnormal presynaptic dopaminergic imaging whilst cases labelled as 'atypical tremor' have also in some instances been associated with an abnormal FP-CIT scan.[23,24]

It remains possible that some of our cases were wrongly categorised by FP-CIT SPECT, rather than clinically mis-interpreted. We consider this unlikely given the 3 year follow up and known sensitivity of FP-CIT SPECT in early PD, and the 100% concordance in distinguishing normal from abnormal imaging results between the two scan assessors.[25]

Video diagnoses in movement disorder patients are constrained by the absence of 'hands-on' testing of rigidity and the subliminal observation of spontaneous movement and body language in real life situations rather than the recording studio. Sometimes several consultations over a number of months may be necessary before a diagnosis can be made with reasonable certainty and historical information may also be a useful distinguishing feature in the differentiation of some patients (e.g. presence of rapid eye movement sleep behavioural disorder or anosmia/hyposmia in PD). Nevertheless both reviewers had identical video material to review, and the findings allow a comparison with studies where similar methods have been used.[26]

This study focussed on the ability of two blinded movement disorder experts to distinguish PD from SWEDDs without the availability of historical details on the patients, and as such tested the capability of step 1 of the QSBCC to distinguish PD from other tremulous disorders. Information on rigidity was unavailable to the video reviewers but increased tone is also a common associated feature of dystonic tremor and Froment's manoeuvre can also elicit cogwheel rigidity in essential tremor.[17,19,20,27]

SPECT imaging of presynaptic dopaminergic pathways have an error rate of around 5% and perhaps greater depending on the expertise of the scanning centre.[28] The diagnostic difficulties in this group of patients are highlighted by the range in sensitivity (72.3-93.3%) and specificity (79.3-85.2%) for the two reviewers, which can be compared with the proportion of SWEDD patients seen in clinical trials which found that 4 to 15% of patients entered as having PD had normal pre-synaptic dopaminergic imaging (although not all of the patients in those studies had a predominantly tremulous presentation).[4, 29,30,] Comparison can also be made with the high error rate in the diagnosis of tremor patients where up to 37% of ET patients were mis-diagnosed as PD (15%) or dystonia (8%).[31]

Analysis of False Negative and Positive Cases

Analysis of the false negative observations is complicated given 6 of 10 patients were on dopaminergic therapy, allowing potential masking of clinical features of PD. This may explain the inability of the examiners to detect bradykinesia but fails to fully explain the examiners' conclusions given that there was only one false negative case shared by the two reviewers.

The difference in diagnosis between the two reviewers is one of the most striking features of the present study (Kappa coefficient = 0.24 95%CI = 0, 0.48). Such variability in clinical opinion is however consistent with studies of inter-rater reliability of Parkinson's disease.[32]

Analysis of the false positive cases in this study shows that interpretation of sequential finger movement testing is the largest contributor to diagnostic error. Despite careful grading of motor slowness, fully-defined bradykinesia (including decremental delay) was observed in 4 SWEDD cases by reviewer 1 and in 11 SWEDD cases by reviewer 2. Possible explanations for this finding could be that bradykinesia occurs in SWEDD patients, despite the absence of substantia nigra pathology or that motor flurries interrupt normal self-paced movement and confound the interpretation.[17] Motor disturbances reminiscent of bradykinesia from the clinical descriptions have also been reported in dystonic tremor .[17] Furthermore, of 30 patients with torticollis reported in 1976, 26 had tremor which was familial in 16, and parkinsonian features were present in 10 including masked facies, bradykinesia and rigidity. Three patients had clinical signs sufficient to entertain a diagnosis of PD.[33]

On the latency of postural tremor in TDPD cases in this series

Re-emergence of postural tremor is felt by some authors to be the postural representation of rest tremor in PD. The latency of the postural or re-emergent tremor in TDPD cases in the current series was not significantly different from the SWEDD cases, which contrasts with a report of a significantly longer latency of postural tremor in PD.[21] However, another study on ET concluded that, “clinically and electrophysiologically the postural tremors of ET and PD are indistinguishable”. [34] Further analysis of larger TDPD cohorts would be worthwhile to validate whether long latency to postural tremor in PD is a differentiating feature from the postural tremor of ET.

Dystonic Features in TDPD patients

It is apparent from this study that a number of false negative cases of untreated PD were felt by the reviewers to have dystonic clinical features such as thumb hyperextension, torticollis or flurries. Retrospective review suggests that isolated thumb hyperextension without associated 'dinner forking' or other dystonic features may be an unreliable sign.

The specificity of clinical features that suggest dystonic tremor may therefore require further consideration.[17] The combination of dystonia and parkinsonism in autosomal recessive parkinsonism and young onset PD is well established.[35,36] Focal dystonia as a feature of PD is also recognised; Lewitt reviewed 10 patients with untreated sporadic PD noting both neck and arm dystonia and dystonic features were also described in a further 9 patients with untreated PD.[37,38]

Conclusions

The accurate diagnosis of predominantly tremulous PD in its early stages remains challenging. The use of FP-CIT SPECT in this study helped us to critically evaluate clinical decision making and to highlight the difficulties in distinguishing TDPD from dystonic tremor, atypical tremor, ET and monosymptomatic rest tremor.

Although the recognition of subtle dystonic features may help re-classify some SWEDD patients as adult dystonic tremor, dystonic features also occur in adult drug naïve PD patients.

A recent note by Bain commented on the difficulty in determining whether there was true bradykinesia in dystonic tremor patients.[39] We consider further motor physiological studies worthwhile to clarify the distinction between the motor abnormalities of dystonic tremor and those considered pathognomonic for parkinsonism.

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Conflicts of Interest

None declared.

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Supplementary Material for On-line Submission

Video Clip. 3 min.

Case 30. False positive diagnosis of PD for reviewer 1 and reviewer 2.

Normal FP-CIT scan.
