Changes in diagnosis with follow-up in an incident cohort of patients with parkinsonism

R Caslake, J N Moore, J C Gordon, C E Harris, C Counsell

ABSTRACT

Background: Accurate diagnosis of the cause of parkinsonism during life can be difficult, particularly at presentation, but few studies have described changes in clinical diagnosis over time and the effect of applying strict research criteria.

Methods: Incident patients with a possible/probable diagnosis of degenerative or vascular parkinsonism had a standardised assessment at diagnosis and at yearly intervals thereafter at which the most likely clinical diagnosis was recorded without strict application of research criteria. Four years after the beginning of the incident period, formal research criteria were applied retrospectively using patient records at baseline and the latest yearly follow-up.

Results: Of 82 incident patients, 66 underwent at least 1 year of follow-up. After a median follow-up of 29 months, clinical diagnosis had changed in 22 (33%). Most (82%) changes occurred in the first year and were due to the development of atypical clinical features, particularly early cognitive impairment; the results of brain imaging; responsiveness to levodopa; and the rate of disease progression. Diagnosis on research criteria differed from latest clinical diagnosis in eight participants (12%). Research criteria gave a "probable" diagnosis in 71% of parkinsonian patients at follow-up but in only 15% at the initial assessment.

Discussion: The clinical diagnosis of the cause of parkinsonism at presentation was often incorrect, even when made by those with a special interest. In particular, Parkinson’s disease was overdiagnosed. Research criteria were often unhelpful in clarifying the diagnosis, even after a median of 29 months of follow-up. Further research is required to identify factors that may be used to improve the accuracy of diagnosis at initial assessment.

Parkinson’s disease (PD) is a neurodegenerative disorder characterised by tremor, rigidity, bradykinesia and postural instability, and associated with a number of non-motor features, including progressive cognitive impairment, neuropsychiatric symptoms, dysautonomia and sleep disturbance.1 Accurate diagnosis of PD is important both in clinical practice, where it will influence management, and in research, where the validity of findings may be compromised if studies include heterogenous conditions. This importance is likely to grow as neuroprotective strategies are developed that target specific pathological processes.

Unfortunately, accurate diagnosis of PD can be difficult. Definitive diagnosis can only be made at post mortem, by demonstration of depletion of brainstem pigmented neurones, with Lewy bodies in the remaining nerve cells.2 Antemortem diagnosis currently relies on clinical assessment but, in
SPECT scans were visually graded by a blinded consultant neuroradiologist as normal, abnormal (graded 1–3)\textsuperscript{14} or atypical (ie, abnormal but not in keeping with the pattern usually seen in neurodegenerative disease).\textsuperscript{23} CT and MRI images were assessed visually for burden of cerebrovascular disease and, in the case of MRI, midbrain atrophy and basal ganglia signal change but no formal criteria were applied.

At baseline and at each yearly follow-up, the assessing (unblinded) clinician recorded up to three most likely clinical diagnoses without applying research criteria and gave a percentage certainty for each (eg, 90% certain PD). Some patients were included in whom the most likely diagnosis was thought to be non-parkinsonian or drug induced parkinsonism, because it was thought that a degenerative or vascular parkinsonian syndrome remained a possibility. All available information was used to inform these diagnoses, including the results of any structural or functional imaging tests that were available at each assessment. Baseline diagnoses were reached after initial clinical assessment, before formal cognitive testing had been carried out.

On each patient’s death, a final assessment was carried out, with review of general practitioner, hospital and research records, imaging tests and taking into account the results of post-mortem examinations where available. A final clinical or pathological diagnosis was then recorded.

In this study, 4 years after the beginning of the incident period, the initial and latest clinical diagnoses (the one with the highest percentage certainty) were compared for all incident patients who had at least 1 year of follow-up data and the reason for any change identified from the notes. In those patients who died, the final clinical diagnosis was taken from the patients’ research records at baseline and latest yearly follow-up.

For 37 of the patients, these criteria were applied independently by two assessors and the diagnoses reached were compared to assess inter-rater reliability. Differences in diagnosis were resolved by discussion. The criteria applied were as follows: the UK Brain Bank criteria for PD,\textsuperscript{3} the consensus criteria for DLB,\textsuperscript{16} the consensus criteria for multiple system atrophy,\textsuperscript{17} the Litvan criteria for progressive supranuclear palsy,\textsuperscript{20} Lees’ proposed criteria for vascular parkinsonism\textsuperscript{19} and the Lang criteria for corticobasal degeneration\textsuperscript{20} disregarding cognitive decline as a criterion for exclusion.\textsuperscript{21} Where patients met more than one set of criteria, a single best fit diagnosis was decided upon by consensus of two authors based on the information available.

### Table 1

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Baseline clinical diagnosis (n (%)</th>
<th>Latest clinical diagnosis (n (%))</th>
<th>Latest diagnosis on research criteria (n (%)</th>
<th>Probable/definite diagnoses only (n = 42) (n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD</td>
<td>46 (70)</td>
<td>37 (56)</td>
<td>32 (48.5)</td>
<td>23 (55)</td>
</tr>
<tr>
<td>DLB</td>
<td>1 (1.5)</td>
<td>7 (10.5)</td>
<td>6 (9)</td>
<td>5 (12)</td>
</tr>
<tr>
<td>Vascular</td>
<td>5 (7.5)</td>
<td>9 (14)</td>
<td>10 (15)</td>
<td>8 (19)</td>
</tr>
<tr>
<td>MSA</td>
<td>4 (6)</td>
<td>3 (4.5)</td>
<td>5 (7.5)</td>
<td>4 (9.5)</td>
</tr>
<tr>
<td>PSP</td>
<td>1 (1.5)</td>
<td>0</td>
<td>2 (3)</td>
<td>2 (4.5)</td>
</tr>
<tr>
<td>CBD</td>
<td>1 (1.5)</td>
<td>1 (1.5)</td>
<td>1 (1.5)</td>
<td>0</td>
</tr>
<tr>
<td>Other: parkinsonian/DIP</td>
<td>3 (4.5)</td>
<td>2 (3)</td>
<td>3 (5)*</td>
<td>N/A</td>
</tr>
<tr>
<td>Other: non-parkinsonian</td>
<td>5 (7.5)</td>
<td>7 (10.5)</td>
<td>7 (10.5)*</td>
<td>N/A</td>
</tr>
</tbody>
</table>

*Patients not meeting any applied research criteria. Parkinsonism defined as two or more of four cardinal motor signs.

CBD, corticobasal degeneration; DIP, drug induced parkinsonism; DLB, dementia with Lewy bodies; MSA, multiple system atrophy; PD, Parkinson’s disease; PSP, progressive supranuclear palsy.

### Table 2

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Between baseline diagnosis and latest clinical diagnosis (n (%))</th>
<th>Between latest clinical diagnosis and research diagnosis (n (%))</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD diagnosis unchanged</td>
<td>33 (50)</td>
<td>32 (48.5)</td>
</tr>
<tr>
<td>Other diagnosis unchanged</td>
<td>11 (16.5)</td>
<td>26 (39.5)</td>
</tr>
<tr>
<td>PD diagnosis changed to other diagnosis</td>
<td>13 (20)</td>
<td>5 (7.5)</td>
</tr>
<tr>
<td>Other diagnosis changed to PD</td>
<td>4 (6)</td>
<td>1 (1.5)</td>
</tr>
<tr>
<td>Other diagnosis changed to other diagnosis</td>
<td>5 (7.5)</td>
<td>2 (3)</td>
</tr>
</tbody>
</table>

PD, Parkinson’s disease.
Table 3  Baseline characteristics of those with and without a change in clinical diagnosis in all patients and in those initially diagnosed with Parkinson’s disease

<table>
<thead>
<tr>
<th>Age (years) (mean (SD))</th>
<th>No change in diagnosis (n = 44)</th>
<th>Change in diagnosis (n = 22)</th>
<th>No change in diagnosis (n = 33)</th>
<th>Change in diagnosis (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Male</td>
<td>74.4 (10.1)</td>
<td>77.5 (6.4)</td>
<td>74.3 (10.4)</td>
<td>76.8 (7.4)</td>
</tr>
<tr>
<td>Symptom duration (months) (median (IQR))</td>
<td>12.7 (6.8–24.2)</td>
<td>12.4 (8.0–20.9)</td>
<td>12.4 (7.2–24.1)</td>
<td>12.2 (8.0–19.2)</td>
</tr>
<tr>
<td>Symptoms described at baseline (n (%))</td>
<td>Tremor 37 (84.1)</td>
<td>17 (77.3)</td>
<td>30 (90.9)</td>
<td>11 (84.6)</td>
</tr>
<tr>
<td></td>
<td>Bradykinesia 31 (70.5)</td>
<td>15 (68.2)</td>
<td>24 (72.7)</td>
<td>10 (76.9)</td>
</tr>
<tr>
<td></td>
<td>Gait disturbance 35 (79.5)</td>
<td>19 (86.4)</td>
<td>25 (75.6)</td>
<td>12 (92.3)</td>
</tr>
<tr>
<td></td>
<td>Postural instability 23 (52.3)</td>
<td>14 (63.6)</td>
<td>15 (45.5)</td>
<td>9 (69.2)</td>
</tr>
<tr>
<td>MMSE (median (IQR))</td>
<td>28 (26–29)</td>
<td>25 (24–28)</td>
<td>28 (26–29)</td>
<td>27 (24–28)</td>
</tr>
<tr>
<td>MMPP (median (IQR))</td>
<td>28 (24–30)</td>
<td>25 (22–28)</td>
<td>29 (26–30)</td>
<td>26 (23–28)</td>
</tr>
<tr>
<td>Motor UPDRS (mean (SD))</td>
<td>25.7 (11.7)</td>
<td>26.4 (8.8)</td>
<td>27.5 (12.1)</td>
<td>24.0 (8.8)</td>
</tr>
<tr>
<td>Total UPDRS (mean (SD))</td>
<td>37.9 (17.3)</td>
<td>38.9 (14.0)</td>
<td>39.5 (18.7)</td>
<td>35.5 (12.5)</td>
</tr>
</tbody>
</table>

MMP, Mini-Mental Parkinson; MMSE, Mini-Mental State Examination; PD, Parkinson's disease; UPDRS Unified Parkinson’s Disease Rating Scale.

Table 4  Reasons for change in clinical diagnosis

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Baseline diagnosis</th>
<th>Revised diagnosis</th>
<th>Reason for change</th>
<th>Patient No</th>
<th>Baseline diagnosis</th>
<th>Revised diagnosis</th>
<th>Reason for change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PD</td>
<td>DLB</td>
<td>Cognitive decline with fluctuating confusion and visual hallucinations in first year.</td>
<td>2</td>
<td>PD</td>
<td>DLB</td>
<td>Early cognitive decline with fluctuating confusion and visual hallucinations.</td>
</tr>
<tr>
<td>2</td>
<td>PD</td>
<td>DLB</td>
<td>Early cognitive decline with fluctuating confusion and visual hallucinations. FP-CIT SPECT scan in keeping with degenerative parkinsonian syndrome.</td>
<td>3</td>
<td>PD</td>
<td>DLB</td>
<td>Early cognitive decline with fluctuating confusion and visual hallucinations.</td>
</tr>
<tr>
<td>4</td>
<td>PD</td>
<td>DLB</td>
<td>Cognitive decline with fluctuating confusion and visual hallucinations.</td>
<td>5</td>
<td>PD</td>
<td>DLB</td>
<td>Cognitive decline with fluctuating confusion and visual hallucinations.</td>
</tr>
<tr>
<td>6</td>
<td>PD</td>
<td>Vascular parkinsonism</td>
<td>Extensor plantar response on examination at review. Multiple vascular risk factors. CT brain scan showed extensive ischaemia.</td>
<td>7</td>
<td>PD</td>
<td>Vascular parkinsonism</td>
<td>No response to levodopa. MRI brain scan showed extensive ischaemia. FP-CIT SPECT scan atypical.*</td>
</tr>
<tr>
<td>8</td>
<td>PD</td>
<td>Drug induced parkinsonism</td>
<td>Levodopa had been started and prochlorperazine stopped before referral. No return of parkinsonism when levodopa stopped.</td>
<td>9</td>
<td>PD</td>
<td>Drug induced parkinsonism</td>
<td>Resolution of parkinsonism on stopping sodium valproate. Normal FP-CIT SPECT scan.</td>
</tr>
<tr>
<td>10</td>
<td>PD</td>
<td>Essential tremor</td>
<td>No progression. No response to levodopa. Development of head tremor. FP-CIT SPECT scan atypical.*</td>
<td>11</td>
<td>PD</td>
<td>Essential tremor</td>
<td>Lack of progression. FP-CIT SPECT scan showed grade 1 abnormality ipsilateral to tremor.</td>
</tr>
<tr>
<td>14</td>
<td>DLB</td>
<td>Vascular parkinsonism</td>
<td>Normal FP-CIT SPECT scan. MRI brain scan showed extensive ischaemia.</td>
<td>15</td>
<td>Vascular parkinsonism</td>
<td>PD</td>
<td>Good response to levodopa. FP-CIT SPECT scan in keeping with degenerative parkinsonian syndrome.</td>
</tr>
<tr>
<td>18</td>
<td>Alzheimer’s associated parkinsonism</td>
<td>DLB</td>
<td>Parkinsonian rest tremor. Fluctuating confusion.</td>
<td>19</td>
<td>Alzheimer’s associated parkinsonism</td>
<td>Vascular parkinsonism</td>
<td>Parkisonian present early in course of dementia. Extensor plantar response on examination. Normal FP-CIT SPECT scan. Structural imaging not performed</td>
</tr>
<tr>
<td>22</td>
<td>Drug induced parkinsonism</td>
<td>Vascular parkinsonism</td>
<td>Residual parkinsonism on withdrawal of prochlorperazine. MRI brain scan showed extensive ischaemia. Normal FP-CIT SPECT scan.</td>
<td>23</td>
<td>Drug induced parkinsonism</td>
<td>Vascular parkinsonism</td>
<td>Residual parkinsonism on withdrawal of prochlorperazine. MRI brain scan showed extensive ischaemia. Normal FP-CIT SPECT scan.</td>
</tr>
</tbody>
</table>

*FP-CIT scan showed punched out lesions not in keeping with the grading system used in degenerative parkinsonian syndromes. These were thought to be a result of cerebrovascular disease.

DLB, dementia with Lewy bodies; FP-CIT SPECT, N-(4-iodophenyl)-(4-carbomethoxy-3)-b-(4-iodophenyl)-b-(4-carbomethoxy-3)-tropane single photon emission computed tomography; MSA, multiple system atrophy; PD, Parkinson’s disease; PSP, progressive supranuclear palsy.

RESULTS

Of 82 incident patients identified, five were excluded from this study because they did not consent to follow-up and 11 were excluded because they died before their first yearly follow-up. The 66 remaining patients had a mean age of 75.0 (SD...
10.5) years at diagnosis and were predominantly male (n = 41 (62%)). At the initial assessment, symptoms had been present for a median of 12.5 months (interquartile range (IQR) 8 to 24). Twenty-four patients died after at least 1 year of follow-up but no other patients were lost to follow-up. Only five patients had undergone examination of the brain at post mortem.

**Change in clinical diagnosis**

Forty-six patients (70%) were initially diagnosed with idiopathic PD, with the most common alternate diagnoses being vascular parkinsonism, multiple system atrophy (all parkinsonian variant) and essential tremor (diagnosed in three patients; table 1). After a median follow-up of 29 months (IQR 23 to 33), the number diagnosed clinically with PD had fallen to 37 (56%), the most common alternative diagnoses being vascular parkinsonism and DLB.

The clinical diagnosis changed between the baseline assessment and the latest follow-up in 22 patients (33%) and remained unchanged in 44 (table 2). There was no significant difference between these two groups in presenting symptoms, severity of their parkinsonian impairment (UPDRS) or duration of their symptoms at baseline (table 3). There was a nonsignificant trend towards greater cognitive impairment (MMSE and MMP) and older age in the group whose diagnoses changed. Most (18, 82%) changes occurred in the first year, three (6% of those with at least 2 years of follow-up) occurred within the second and one (5% of those with at least 5 years of follow-up) within the third.

In those initially diagnosed with PD, the diagnosis was most likely to change to DLB (5/13, 38%) or essential tremor (3/13, 23%) (table 4). Changes were most commonly a result of development of additional features (n = 7), particularly early cognitive impairment and neuropsychiatric features (n = 5), the results of radiological imaging (n = 6), poor response to levodopa (n = 4) and lack of disease progression (n = 6).

Changes in diagnosis in those initially diagnosed with conditions other than PD are shown in table 4. Changes were to PD (4/9, 44%), vascular parkinsonism (5/9, 55%) or DLB (2/9, 22%). The predominant reason for change to a diagnosis of PD was response to levodopa. In the two patients whose diagnoses changed from essential tremor to PD, both had FP-CIT SPECT scans that were abnormal in a pattern not in keeping with the usual pattern in neurodegenerative disorders and were reported as being likely to be indicative of cerebrovascular disease. Clinically, these patients were not felt likely to suffer from vascular parkinsonism. All three patients whose diagnoses changed to vascular parkinsonism had normal FP-CIT SPECT scans.

**Differences between latest clinical diagnosis and research diagnosis**

Agreement between the two observers on the research criteria diagnosis was good (κ = 0.73), and agreement between research diagnosis and clinical diagnosis was very good (κ = 0.82). Details of the eight participants whose clinical and research diagnoses differed are shown in table 5.

When research criteria were applied using only the information available at baseline, 46 patients had a “possible” diagnosis according to the criteria, 10 had a “probable” diagnosis and 10 could not be diagnosed using the criteria applied. Where the most up to date information was used, 10 patients could not be diagnosed (three had an unspecified parkinsonian syndrome and seven were not parkinsonian), 14 had a “possible” diagnosis, 37 had a “probable” diagnosis and five had a “definite” diagnosis (table 1). Of the 14 patients whose diagnosis by research criteria was rated “possible”, six (45%) also met the criteria for “possible” diagnosis of another syndrome.

In the five patients who underwent post-mortem examination, the final clinical, research and pathological diagnoses agreed in two (one PD and one vascular) and in one the clinical diagnosis and pathological diagnosis agreed (PD with coexistent Alzheimer’s disease) while research criteria suggested possible DLB. The two participants whose clinical and antemortem research diagnoses were PD but whose post-mortem examinations showed PSP are described in table 5.

**DISCUSSION**

In this study, one-third of the initial diagnoses of the cause of parkinsonism changed over a median of 29 months of follow-up. The majority of this change was a result of initial...
overdiagnosis of PD. The proportion of patients diagnosed with PD fell by 14% and most (59%) diagnostic changes were away from PD. These results are similar to previous studies, which showed that the clinical diagnosis of parkinsonism changed over time in 36% of patients attending a highly specialist clinic, and 16% of an incident cohort of patients with PD according to UK Brain Bank criteria had their initial diagnosis changed after about 3.5 years of follow-up.

Over one-third (38%) of those misdiagnosed with PD had their diagnosis changed to DLB. This was usually because the symptomatic cognitive features of the disease were absent initially as, even using the strictest interpretation of the diagnostic criteria, parkinsonism can predate the onset of dementia by up to 1 year. However, the suggestion of a difference in cognitive scores between the change and no change groups would suggest that there may have been detectable deficits despite the lack of cognitive symptoms. The MMSE and MMF may, therefore, be of some use in identifying parkinsonian patients who will go on to develop early dementia, although a larger study is needed to test this hypothesis.

Essential tremor was clinically misdiagnosed as PD in three of our patients, which is not unexpected. While an essential tremor is characteristically more prominent on posture holding and action, it can also cause a tremor at rest and may even be associated with rigidity. Conversely, PD may present with an isolated asymmetric postural tremor in the absence of other features and may only cause other features of parkinsonism after many years.

Changes in diagnosis were usually as a result of level of response to treatment with levodopa or a dopamine agonist, lack of progression and development or resolution of atypical clinical features. This serves to highlight the need for regular follow-up and diagnostic revision in parkinsonian patients, and the diagnostic value of a trial of therapy. However, the results of therapeutic trials must be interpreted with caution, as PD may not show the classical excellent response to treatment, and there have been reports of pathologically established cases of PD without an appreciable response to levodopa, making clinical diagnosis in life extremely difficult. The role of functional imaging in the changes to diagnosis that took place is unclear. The results of FP-CIT SPECT scans supported change from initial clinical diagnoses in six patients, although in all but one of these cases (patient No 14, table 4), the clinical features were present that may have led to the change independently. In the two changes in diagnosis from essential tremor to PD, the FP-CIT SPECT scan results were not typical of a neurodegenerative disorder.

The latest clinical diagnosis differed from the diagnosis on research criteria in eight patients (12%; table 5) usually because of exclusion criteria within the research criteria (patient Nos 23 and 24), or arbitrary time limits within the criteria (patient Nos 28 and 29). In two cases (patient Nos 25 and 30) there were features to support both diagnoses and it could be argued that either could be applied. In two patients (patient Nos 26 and 27), whose pathological diagnosis differed from their research and latest clinical diagnosis, PSP was mistaken for PD. It is increasingly recognised that, as well as causing the typical syndrome of postural instability, supranuclear gaze palsy and cognitive dysfunction (Richardson’s syndrome), PSP can present with an asymmetric parkinsonian syndrome very similar to PD, although with a blunted response to dopaminergic therapy (PSP parkinsonism).

Research criteria were of limited value in supporting a clinical diagnosis in parkinsonian patients. At baseline assessment, most (70%) diagnoses were rated “possible” and only 10 patients (15%) met the criteria for “probable” diagnosis of a parkinsonian syndrome. Even after about 2.5 years of follow-up, a significant proportion of diagnoses in those with a parkinsonian disorder remained unclassifiable (5%) or “possible” (24%) and nearly half of the latter met criteria for another “possible” diagnosis.

The main strength of our study was that it involved a community-based incident cohort where steps were taken to identify as high a proportion of parkinsonian patients as possible, standardised prospective assessments were made each year by a specialist with an interest in movement disorders and few patients were lost to follow-up. The patients are, therefore, likely to be representative of parkinsonian patients in general, and not only those usually seen at specialist clinics, while the prospective data collection allowed accurate application of the research criteria.

However, there are also some limitations of this study that are worth noting. Firstly, our cohort was relatively small as it was drawn from a pilot study. A larger incidence study is currently underway and will allow analysis of similar data in a larger population. Secondly, while the revised diagnoses here are taken to be correct, it is likely that they will continue to change over time. Follow-up of this cohort will continue and so it will be possible to report the pattern of changes with longer follow-up in the future. Thirdly, the diagnoses were all supervised by a single consultant with an interest in PD and so our results may not be generalisable, particularly to more generalist clinics where the misdiagnosis rate may be higher. Finally and importantly, few diagnoses have been confirmed by post mortem and, therefore, it is not clear whether either the clinical or antemortem research diagnoses are correct. This is a problem with all clinical studies of parkinsonism but, because we have systematically tried to approach our participants for antemortem consent, we hope to obtain pathological confirmation in more of our participants over time. Studies of diagnostic accuracy that used brain bank material had the advantage of complete pathological confirmation but were disadvantaged by limited clinical information on which to base research diagnostic criteria and limited generalisability because post mortems are often performed on unusual or difficult cases.

The clinical significance of misdiagnoses in parkinsonism varies. A change in diagnosis between a parkinsonian condition and a non-parkinsonian one (such as essential tremor) will have a significant impact on patient care as the treatments are quite different. Changing between different parkinsonian syndromes may not alter management so dramatically as a trial of dopamine replacement therapy is often warranted but it will alter what information is given to the patient about prognosis. In addition, some clinicians may wish to avoid early levodopa in those with PD because of the risk of motor complications, while treatment withdrawal should always be considered where the syndrome is thought to be unresponsive. Similarly, while many experts would regard PD and DLB as being part of the same disease spectrum, we regarded them separately because their prognosis differs and dopamine agonists may be less suitable in DLB because of their greater neuropsychiatric toxicity.

In summary, we have demonstrated that even in those with a special interest, the accurate diagnosis of the cause of parkinsonism at presentation was difficult, that PD was overdiagnosed at first assessment and that research criteria were often unhelpful in clarifying the diagnosis, even after a median of 29 months of follow-up. Further research, in larger groups and over longer periods, is necessary to identify factors
that may be used to improve the accuracy of diagnosis at the initial assessment. Our findings support the recent NICE guidelines that regular clinical review of those suffering from parkinsonism with careful attention to the diagnosis is essential in order that they receive appropriate care.30

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REFERENCES

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