

Depression and voice handicap in Parkinson disease



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ABSTRACT

Background: Dysphonia is common in Parkinson's disease (PD), but the mechanism underlying the development remains unclear. This study investigated possible clinical factors related to PD dysphonia.

Methods: Dysphonia severity was assessed in 147 non-demented patients with PD and 30 non-PD controls using the Voice Handicap Index (VHI)-10. A threshold of 12 VHI-10 score was used to define the presence of dysphonia. The severity of PD and depression was measured using the Unified Parkinson's Disease Rating Scale (UPDRS)-motor and the Geriatric Depression Scale (GDS).

Results: Dysphonia was observed in 52 patients (35.4%). Patients with dysphonia scored higher on the UPDRS and GDS scores compared with patients without dysphonia. Multivariate logistic regression analysis revealed that GDS was the only factor significantly associated with the presence of dysphonia. Non-PD controls did not show this association.

Conclusions: The results support a high prevalence of dysphonia in patients with PD, and suggest that the presence of dysphonia is more closely related to depression than to the severity of PD.

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Introduction

Dysphonia frequently accompanies Parkinson disease (PD), and up to 70% of patients with PD experience impaired speech during the disease process [1]. PD dysphonia is characterized by a monotony of pitch and loudness, reduced stress, variable rate, short rushes of speech, imprecise consonants, and a breathy and harsh voice [2,3]. Dysphonia can appear at any stage of PD, although it usually worsens in the later stage [2]. Orofacial-laryngeal bradykinesia and rigidity have been suggested as contributors to PD dysphonia [2,3], but the effects of dopamine replacement and deep brain stimulation remain controversial [2]. Additionally, the relationship between changes in voice parameters and PD severity is limited [4]. These observations suggest that voice handicaps associated with PD may not result simply from dopaminergic depletion, but may also be related to non-dopaminergic lesions.

Approaches to the assessment and treatment of voice disorders associated with PD have focused primarily on objective voice measurements (i.e., acoustic and aerodynamic measures of voice), and relatively few patients are assessed and treated for voice disorders [4–8]. Although objective measures can detect subtle changes in each component of

voice production, they do not address global vocal functioning from the patient's perspective [9]. The Voice Handicap Index (VHI)-10, a shortened version of the VHI, is a voice disorder-specific, patient-based assessment tool that can assess the impact of the voice problems on the patient's overall daily functioning [9]. It is possible that the VHI-10 may provide more information about PD dysphonia than do measurements of the biological and physiological variables associated with voice production. In this study, we measured the VHI-10 in patients with PD and investigated factors related to voice handicaps in this population.

1. Patients and methods

We recruited consecutive patients with PD from the outpatient clinic of our hospital. The diagnosis of PD was made according to the UK Brain Bank Criteria [10]. Patients were excluded if they had atypical or secondary parkinsonism, scored 23 or less on the Mini-Mental Status Examination (MMSE), or had focal neurological deficits other than parkinsonism. This study was approved by the local ethics committee.

Dysphonia was assessed using the VHI-10, which consists of 10 items (5 physical, 4 emotional and 1 functional item) rated from 0 (never) to 4 (always), with higher scores indicating increasing severity of voice disability [9]. The presence of dysphonia was defined as a VHI-10 score of 12 or higher [11]. Geriatric Depression Scale (GDS)-15 was used to assess depression [12].

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We collected demographic and clinical data about the participants, and assessed each patient's disease severity using the Unified Parkinson's Disease Rating Scale (UPDRS). The following items from the UPDRS-motor were used in the analysis of each motor symptom; items 20 and 21 for tremor score, 22 for rigidity score, 23–26 and 31 for bradykinesia score, and 27–29 and 30 for the postural/gait score. To minimize the influence of speech dysfunction on the assessment of PD severity, the UPDRS-motor score, excluding item 18 (speech function), was used. Based on methods previously described [13], we classified patients into tremor-dominant and postural instability/gait difficulty (PIGD) types. The levodopa equivalent dose was calculated as described previously [14].

After the analysis of the PD patients' data, we measured VHI-10 and GDS in 30 age-matched patients who visited our clinic because of chronic headache, in order to investigate the relationship between depression and voice handicap in non-PD controls.

Data are expressed as means $\pm$ SDs. Clinical data for patients with and without dysphonia were compared using independent *t*-tests (for numeric data) and chi-square tests (for non-numeric data). A multivariate logistic regression analysis was performed to identify factors significantly associated with the presence of dysphonia. The performance of the proposed regression model was evaluated with a receiver operating characteristic (ROC) curve. SAS (v 9.2) for Windows (SAS Institute, Cary, NC, USA) was used to perform all statistical analyses, and $p < 0.05$ was considered significant.

2. Results

In total, 147 patients (age, 65.6 ± 8.9 yrs; 64 men) were recruited: of these, 52 patients (35.4%) were diagnosed with PD dysphonia. Clinical data between the patients with and without dysphonia are shown in Table 1. Patients with dysphonia showed significantly higher GDS and UPDRS-motor scores compared with those without dysphonia, whereas age, gender distribution, PD duration, levodopa equivalent dose, and MMSE scores were comparable between the two groups. The analyses of each motor symptom revealed that patients with dysphonia had significantly higher rigidity and posture/gait scores compared with patients without dysphonia, but the two groups were comparable with respect to tremor and bradykinesia scores. Overall, 41 patients (28.0%) were classified as tremor-dominant type, and 96 patients (65.2%) were PIGD type. The mean VHI-10 scores of the tremor-dominant (11.5 ± 11.5) and PIGD (10.9 ± 10.0) groups were comparable.

To investigate the factors contributing to the presence of PD dysphonia, we performed a multivariate regression analysis with the presence of dysphonia as the dependent variable, and age, gender, PD duration, UPDRS-motor and GDS scores as the independent variables. This analysis revealed that GDS score (odds ratio (OR), 1.253, 95% confidence interval (CI), 1.125–1.396, $p < 0.001$) was the only significant factor

associated with the presence of dysphonia. The area under the ROC curve for this regression model was 0.773 (95% CI, 0.697–0.848) (Fig. 1A). Although patients with dysphonia showed significantly higher UPDRS-motor score than those without dysphonia, significance of this association disappeared after adjusting for the influence of other variables (OR, 1.004, 95% CI, 0.980–1.051, $p = 0.856$). GDS score was significantly correlated with VHI-10 score ($r = 0.372$, $p < 0.001$, Fig. 1B) after adjustment of the influence of age, gender, PD duration and UPDRS-motor score. GDS score was also significantly correlated with VHI-10 score after excluding four items in the emotional domain ($r = 0.383$, $p < 0.001$).

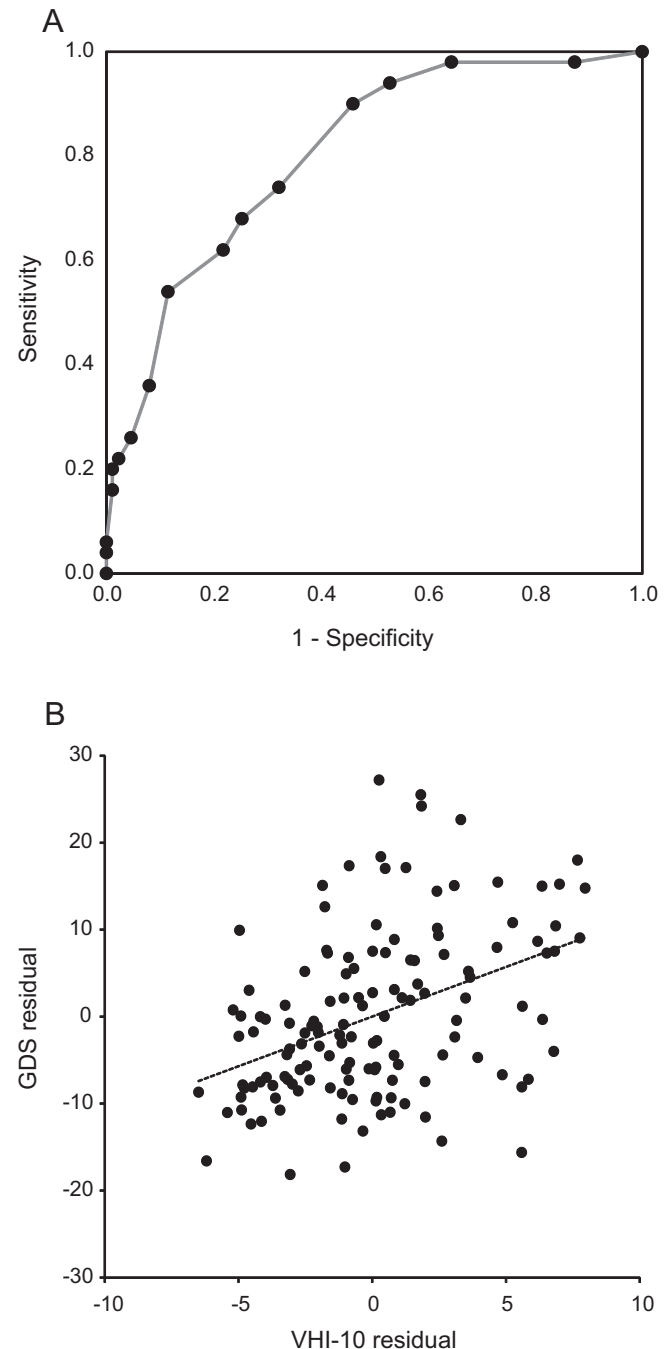


Fig. 1. (A) The receiver operating characteristic curve for predicting the presence of dysphonia in a logistic regression model including GDS score as an independent variable. (B) The scatterplot showing a significant correlation between GDS and VHI-10 scores after adjustment of the influence of other variables including age, gender, PD duration and UPDRS-motor score.

Table 1
Clinical data between the patients with and without dysphonia.

Variables	Dysphonia (–) (n = 87)	Dysphonia (+) (n = 50)	p-Value
Age (yrs)	64.4 $\pm$ 8.4	65.7 $\pm$ 9.2	NS
Gender (% women)	60%	52.9%	NS
PD duration (yrs)	3.5 $\pm$ 3.4	4.5 $\pm$ 3.1	NS
L-Dopa equivalent dose (mg)	522 $\pm$ 272	572 $\pm$ 237	NS
MMSE	26.8 $\pm$ 2.7	26.1 $\pm$ 3.0	NS
GDS	3.6 $\pm$ 3.1	7.7 $\pm$ 3.7	<0.001
UPDRS-motor	18.1 $\pm$ 7.9	23.3 $\pm$ 9.4	0.001
Tremor	2.1 $\pm$ 1.6	2.4 $\pm$ 1.9	NS
Bradykinesia	8.1 $\pm$ 4.1	9.5 $\pm$ 3.9	NS
Rigidity	2.5 $\pm$ 2.6	4.1 $\pm$ 3.3	0.003
Posture/gait	4.6 $\pm$ 2.1	6.4 $\pm$ 4.0	0.001

PD, Parkinson disease; MMSE, Mini-Mental Status Examination; GDS, Geriatric Depression Scale; UPDRS, Unified Parkinson's Disease Rating Scale; NS, not significant.

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