Prevalence and Characteristics of Alexithymia in Parkinson's Disease

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Background: *Alexithymia, a reduction in the tendency to think about emotions, together with a difficulty in identifying and describing feelings, has been characterized as a personality trait, but may be secondary to other pathological conditions.* **Objective:** *The authors aimed at investigating alexithymia in Parkinson's disease (PD).* **Method:** *Seventy PD patients and 70 control subjects were administered the 20-item Toronto Alexithymia Scale.* **Results:** *The authors found that 21.4% of PD patients and 10.0% of controls could be classified as alexithymic. PD patients and controls significantly differed on global levels of alexithymia. However, univariate analyses showed that PD patients differed significantly only on the subscale investigating difficulty describing and communicating feelings.* **Conclusion:** *These results indicate that some facets of alexithymia are a relevant feature of PD, possibly in relation to the neuropathological changes that characterize the disease.* (Psychosomatics 2010; 51:22–28)

Alexithymia is a cognitive-affective disturbance mainly characterized by a relevant reduction in the tendency to think about emotions together with a difficulty in identifying and describing feelings and distinguishing feelings from bodily sensations of emotional arousal.[1] According to some authors,[2] alexithymia could be a personality trait that contributes to the development and severity of somatic and psychopathological disorders.[3–5] Results from other studies suggest, instead, that alexithymia may be secondary to other pathological conditions.[6–8]

There are few reports in the literature on alexithymia in individuals with Parkinson's disease (PD). Nevertheless, some studies indicate that an impairment in the processing of emotional experience may be present in PD patients.[9–14] (However, Adolphs et al.[15] present a partially discrepant view.) In particular, Jacobs et al.[11] showed impaired performance in PD patients on a task assessing emotional facial imagery and on tasks probing emotional expression and the perception of emotion in faces. Recently, Dujardin et al.[16] described impaired decoding of facial emotions in PD patients at an earlier stage of the disease. Moreover, Simons et al.[17] reported PD patients' specific difficulty, as compared with healthy-control subjects, in creating emotional facial expressions. Results consistent with PD patients' deficit in communicating and decoding emotions were obtained in studies on the ability to recognize and express affective verbal expression.[9–11]

In a previous study, we found that alexithymia occurred in about 21% of a cohort of 58 PD patients and that it was strongly associated with severity of depression.[18] We speculated that a dysregulation of the dopaminergic mesocorticolimbic system, which occurs in PD and is reported to be more severe in PD patients with depression,[19] could be related to the described alexithymic ex-

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pression. However, in that study, the lack of a control sample without PD prevented us from drawing firm conclusions about the prevalence of alexithymia in PD and whether the association between alexithymia and depression is particularly strong in PD or simply reflects an overlap of the depressive and alexithymic symptoms widely documented in persons without PD.[20]

The present study was aimed at investigating the prevalence and characteristics of alexithymia in hospitalized PD patients without dementia, as compared with a group of hospitalized control subjects suffering from orthopedic and peripheral nervous system pathologies. We also investigated the relationship between alexithymia and depressive and anxious symptoms in PD versus brain damage-free control groups.

METHOD

Subjects

After giving their informed consent, 70 PD patients (45 men and 25 women) and 70 control subjects (CS; 42 men and 28 women) consecutively admitted to a neurorehabilitation hospital participated in the study. PD patients suffered from a mild-to-moderate rigid/akinetic form of idiopathic PD and were hospitalized to adjust antiparkinsonian medication and undergo a motor-rehabilitation program. The diagnosis of idiopathic PD was made by an expert neurologist on the basis of 1) the presence of at least two of the four cardinal parkinsonian symptoms; and 2) good chronic response to *L*-dopa treatment. Exclusion criteria included the following: 1) dementia suspected on the basis of clinical examination or Mini-Mental State Exam (MMSE) score $\leq$24;[21] 2) presence of severe systemic and metabolic disease (such as, diabetes, hypothyroidism, etc.); 3) marked cortical and subcortical atrophy and/or ischemic vascular lesions on computed tomography (CT) and/or MRI scans; 4) history of neurological disorders other than PD; 5) evidence of psychotic symptoms; and 6) severe functional impairment of the autonomic nervous system. PD patients were clinically evaluated with the Unified Parkinson's Disease Rating Scale (UPDRS), Part 3.[22]

Extrapyramidal symptoms predominantly affected the right side in 34 PD patients and the left side in the remaining 36 patients. All patients were treated with L-dopa and/or a dopaminergic agonist; 25 PD patients were also treated with antidepressant medication. Moreover, 36 of 70 showed an unstable response to therapy, characterized by reduction of efficacy of single dose (Wearing Off) and Abnormal Involuntary Movements (AIMs) of peak dose, and therefore presented a long-term treatment syndrome (LTTS). On the other hand, 34 patients showed a stable and consistent therapeutic response to treatment. All patients were evaluated in "on" clinical status, about 30 minutes after the first daily drug administration, also considered as the best "on" condition in fluctuating patients.

The CS group comprised patients suffering from orthopedic diseases (e.g., limb fractures, traumatic limb amputation, hip prosthesis; N=57) or peripheral nervous system pathologies (e.g., polyneuropathy, radiculoneuropathy; N=13) who were also admitted to our hospital to undergo a physical rehabilitation program. Exclusion criteria for the CS group included: 1) dementia, suspected on the basis of clinical examination, or an MMSE score $\leq$ 24; 2) presence of severe systemic or metabolic disease; 3) intake of medication active in the CNS or with CNS side effects; and 4) history of psychiatric or neurological illness, head trauma, or substance abuse. Both PD patients and control participants were tested within 2 weeks of hospitalization. Demographic and clinical characteristics of the two groups are reported in Table 1. The study was approved by the local ethics committee.

Psychopathological Evaluation

Alexithymia was assessed by the 20-item Toronto Alexithymia Scale (TAS–20),[23,24] an extensively validated self-report questionnaire. The scale comprises three subscales that investigate the following factors: F1) difficulty identifying feelings (e.g., "I am often confused about what emotion I am feeling;" "I have feelings that I cannot quite identify."); F2) difficulty describing and communicating feelings (e.g., "I find it hard to describe how I feel about people;" "It is difficult for me to reveal my innermost feelings, even to close friends."); F3) externally-oriented thinking (e.g., "I prefer to analyze problems rather than just describe them;" "I prefer talking to people about their daily activities rather than their feelings."). The total score on the questionnaire allows us to categorize subjects as non-alexithymic (score: 20–51), borderline alexithymic (score: 52–60), or alexithymic (score $\geq$61). Severity of depression was evaluated with the Beck Depression Inventory (BDI),[25] which is considered a reliable instrument for examining severity of depression in PD.[26] To control for the effect of PD somatic symptoms on the overall depression score, we considered separately in the statistical analyses the partial scores on cognitive-affective (BDI–Psy; e.g., "feel said and disappointed," "feel like a