SHORT REPORT

Obsessive-compulsive symptoms in Parkinson's disease

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Abstract

To systematically investigate obsessive-compulsive traits in Parkinson's disease, patients were administered the Maudsley obsessional-compulsive inventory (MOCI) and a modification of the Leyton obsessional inventory (LOI) to a sample of non-demented and non-depressed patients with Parkinson's disease. Patients with severe Parkinson's disease showed more obsessive traits than normal controls in MOCI and LOI total scores, and in the "checking", "doubting", and "cleaning" subscales of the MOCI. By contrast, patients with mild disease did not differ from controls. A significant correlation was found between severity and duration of illness and MOCI total score. These results support the involvement of basal ganglia in obsessive-compulsive symptomatology. As patients with mild Parkinson's disease did not differ from controls, obsessive-compulsive disorder does not seem to be directly related to the initial nigrostriatal dopaminergic deficiency which causes clinical Parkinson's disease symptomatology. The appearance of obsessive symptoms could be related to the subset of neurochemical changes taking place at the level of the basal ganglia circuitry as disease progresses.

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Several structural and functional neuroimaging studies have shown that obsessive-compulsive disorder is related to dysfunction of the basal ganglia.[1][2] Lesions in the basal ganglia produce obsessive-compulsive symtomatology similar to idiopathic obsessive-compulsive disorder.[3] In addition, the incidence of obsessive-compulsive disorder is high in Huntington's disease and Tourette's syndrome.[4–6]

It is well known that patients with Parkinson's disease manifest several dysfunctions of frontobasal ganglia circuitry. Dysfunction in the limbic circuitry may also be responsible for the occurrence of obsessive-compulsive traits in patients with Parkinson's disease. Tomer *et al*[7] found a relation between obsessive-compulsive symptomatology and the severity of motor impairment in Parkinson's disease.

However, in a group of 16 patients with Parkinson's disease Müller *et al*[8] did not find differences between patients and normal controls in the Maudsley obsessional-compulsive inventory (MOCI). In a previous study, we reported that patients with Parkinson's disease who had undergone pallidotomy had improved scores on the MOCI.[9]

The aim of the present study was to systematically investigate obsessive-compulsive traits in Parkinson's disease. Two questionnaires were used: the MOCI, mainly applied in idiopathic obsessive-compulsive disorder,[10] and a modified version of Leyton obsessional inventory (LOI), which was reported to be sensitive to Tourette's syndrome.[6]

Methods

SUBJECTS

The MOCI was administered to 72 consecutive non-demented patients with idiopathic Parkinson's disease (36 men and 36 women) from the Department of Neurology at the Hospital Clinic Universitari in Barcelona. The control group comprised 72 subjects without history of neurological or psychiatric illness (36 men and 36 women). They were patients' spouses or friends, recruited from the neurology outpatients' department of the Hospital Clinic Universitari. The groups were matched by age, sex, and education. The mean (SD) age of the patients was 63.25 (8.58), educational level 8.56 (4.32) years; for controls, the mean (SD) age was 63.63 (10.13), educational level 9.39 (4.31) years. Mean (SD) age at onset of Parkinson's disease was 51.90 (10.65) years, duration of disease 11.51 (7.53) years, and mean Hoehn and Yahr stage 3.03 (SD 1.12).

A modified version of the LOI was administered to a subgroup of 54 patients with Parkinson's disease (27 men, 27 women) and 54 normal controls (27 men, 27 women) from the sample. Mean (SD) patient age was 64.13 years (8.91), educational level 8.85 (4.44) years, duration of illness 10.65 (8.00) years, and mean Hoehn and Yahr stage 2.91 (1.19). The control group was also matched to the Parkinson's disease group by mean (SD) age (64.15 (10.51)), education (9.39 (4.45) years), and sex (27 men, 27 women).

Comparison between mild Parkinson's disease (PD), severe PD, and control groups in MOCI and LOI questionnaires

	Mild PD	Severe PD	Controls	F Value	Post hoc
Group 1:	(n=25)	(n=47)	(n=72)	(2,141)	
MOCI total	4.12 (3.15)	7.62 (4.19)	4.83 (3.57)	10.412***	†‡
MOCI checking	1.08 (1.29)	2.23 (2.01)	1.26 (1.74)	5.326**	‡
MOCI cleaning	1.28 (1.43)	1.87 (1.41)	1.21 (1.37)	3.417*	†‡
MOCI doubting	1.48 (1.36)	3.09 (1.60)	2.18 (1.48)	10.331***	†‡
MOCI slowness	2.28 (0.61)	2.32 (1.07)	2.14 (0.74)	0.724	
Group 2:	(n=23)	(n=31)	(n=54)	(2,105)	
LOI	48.04 (29.04)	62.03 (28.18)	48.41 (22.75)	3.14*	†‡
LOI TS items	1.30 (2.18)	2.03 (3.05)	1.44 (1.79)	0.86	
LOI OCD items	16.43 (11.55)	21.06 (9.27)	18.31 (8.37)	1.70	

Values are means (SD).
*p<0.05; **p<0.01; ***p<0.001 from controls.
†Significant differences between mild PD and severe PD.
‡Significant differences between severe PD and normal controls.
TS=Tourette's syndrome; OCD=obsessive-compulsive disorder.

ASSESSMENT OF OBSESSIVE-COMPULSIVE TRAITS

The MOCI comprises 30 sentences which are classified into four factors: checking (nine items), cleaning (11 items), doubting (seven items), and slowness (seven items). Subjects were instructed to answer true or false. The inventory includes both affirmative and negative sentences.

The modified version of the LOI, proposed by Frankel *et al*,[6] was constructed from questions derived from the original LOI,[11] supplemented by additional questions designed to elicit obsessive-compulsive disorder phenomena. The LOI consists of 40 statements, and subjects were asked to make a graded response indicating whether the statement was true for his or her symptoms: "never" (score 0), "rarely" (1), "sometimes" (2), "moderately often" (3), "frequently" (4) or "always" (5). Frankel *et al*[6] identified a group of items (14, 19, 21, 24, 29, 38, and 40) as preferentially endorsed by patients with Tourette's syndrome and 11 questions (9 to 12, 15, 23, 25, 28, 30, 34, and 37) that elicited high scores from patients with idiopathic obsessive-compulsive disorder.

Results

Patients with Parkinson's disease obtained a higher total MOCI score than normal controls (t=2.42, p=0.017). As MOCI total score correlated with Hoehn and Yahr stage (r=0.33, p=0.004) and duration of illness (r=0.34, p=0.005), two subgroups were created according to severity of Parkinson's disease: mild (Hoehn and Yahr≤2.5) and severe (Hoehn and Yahr>2.5). One way analysis of variance (ANOVA) showed a significant effect of group (F=10.41, p=0.0001). Post hoc analysis using Duncan's procedure showed higher scores in patients with severe Parkinson's disease than in those with mild Parkinson's disease and normal controls (table). The analysis of MOCI factors showed a significant group effect on the "checking", "doubting" and "cleaning" subscales (analysis of variance (ANOVA), F=5.33, p=0.0059; F=10.33, p=0.0001; and F=3.42, p=0.0355, respectively).

The LOI scores also disclosed significant differences between groups. The obsessional scores for patients with severe Parkinson's disease were higher than those for patients with mild Parkinson's disease and controls. Taking a cut off level of 70 to determine a definitely raised inventory score,[6] 13 of the 54 patients (24.1%) and 10 of the 54 control subjects (18.5%) scored in this range. Total scores for MOCI and LOI were strongly correlated in both patients with Parkinson's disease (r=0.70, p=0.000) and controls (r=0.65, p=0.000). Leyton total score was not related to age (r=0.15, p=0.288), Hoehn and Yahr (r=0.02, p=0.885) or duration of illness (r=0.05, p=0.729).

Discussion

Relations between Parkinson's disease and obsessive-compulsive disorder have been previously suggested in other studies,[12][13] but few researchers have systematically investigated this contention. Hardie *et al*[13] described complex mannerisms and organised rituals in conjunction with the on-off phenomenon in patients with Parkinson's disease, but only two studies have hitherto administered obsessive-compulsive questionnaires. Tomer *et al*[7] compared data from 30 patients with Parkinson's disease with the normative data from Leyton's original questionnaire. They found that out of 30 patients, 17 had a higher symptom score, and 19 a higher trait score than the mean of normal controls. The authors concluded that obsessive-compulsive symptoms may be an important but unrecognised feature in some patients with idiopathic Parkinson's disease. Müller *et al*[8] reported negative results using the MOCI and the Hamburg obsessive-compulsive inventory in a sample of 20 patients with Parkinson's disease and 43 controls. The small sample size and the heterogeneity of Parkinson's disease may be responsible for the lack of statistical significance in this study.

Patients with severe Parkinson's disease presented significantly more self reported obsessive-compulsive symptoms than controls in both questionnaires administered, but none of the patients was diagnosed as having obsessive-compulsive disorder according to DSM-IV criteria, and none were receiving psychopharmacological treatment for obsessive-compulsive disorder symptomatology. Although the MOCI and LOI scales are widely accepted as descriptive self estimation scales for obsessive-compulsive symptoms, no diagnostic value is given.

Our patients with severe Parkinson's disease differed from controls in almost all scales (checking, cleaning, and doubting). Patients with mild Parkinson's disease had no obsessive-compulsive symptoms. In addition, a correlation between years of evolution and MOCI global score was found. Thus, the present study showed that obsessive-compulsive symptoms appeared late during the disease progression in patients with idiopathic Parkinson's disease. This fact suggests that the emergence of obsessive symptoms could be directly related to the subset of neurochemical changes taking place at the level of the basal ganglia circuitry as the disease progresses.[14] The functional disturbances produced by degeneration of the nigrostriatal pathway could influence the striatofrontal circuits in the advanced stages of

Parkinson's disease. Another alternative explanation could be that some of the patients in our study presented direct frontocortical damage—that is, gliosis, neuronal loss, and Lewy bodies in the cytoplasm. However, this explanation is unlikely as the patients involved in this study showed no signs of dementia, hallucinations, or any other symptomatology of Lewy body dementia.[15]

Obsessive-compulsive disorder is mainly seen in degenerative processes such as Huntington's disease[4] and is associated with Tourette's syndrome.[5] The motor symptoms of Parkinson's disease are in some ways the opposite to Tourette's syndrome and Huntington's disease, as in these two diseases there is a motor overactivation. However, over time levodopa treatment in Parkinson's disease is able to induce dyskinesias. In patients with very advanced Parkinson's disease who underwent pallidotomy we found an improvement of both obsessive-compulsive traits and dyskinesias in the assessment performed 3 months after surgery,[9] suggesting that their mechanisms are similar. Litvan *et al*[16] found that hyperkinetic syndromes, such as Huntington's disease, are associated with hyperactive behaviours. They suggested that these behaviours are secondary to an excitatory subcortical output through the medial and orbitofrontal cortical circuits.

Several models of obsessive-compulsive disorder—and other psychiatric diseases such as melancholia—centre around the possibility that symptoms represent behavioural programmes that are pathologically and repetitively generated by dysfunctional basal ganglia circuits, as in motor dysfunction.[17][18] Lesional data in Parkinson's disease also point to a link between obsessive-compulsive disorder and basal ganglia. Daniele *et al*[19] reported a case of idiopathic Parkinson's disease which started obsessive thoughts and compulsions after a vascular unilateral lesion restricted to the left putamen. Obsessions appeared after an interval of days, compulsions after a few weeks, and both symptoms progressed over subsequent months. This patient was classified as very severe on the Yale-Brown obsessive-compulsive scale.

Analysing the items that Frankel *et al*[6] identified as preferentially endorsed by patients with Tourette's syndrome, we did not find differences between patients with Parkinson's disease and controls. The classic obsessive-compulsive disorder symptoms found in Tourette's syndrome (questions which involved blurting obscenities, imitating the movements of others, counting compulsions, and impulses to hurt oneself) were not found in our Parkinson's disease sample. Slowness might be expected to be characteristic of Parkinson's disease from a clinical point of view, but the MOCI score on this subscale (slowness and repetition) did not increase.

It has been suggested that patients with Parkinson's disease display a specific cluster of personality traits consisting of increased rigidity, conscientiousness, industriousness, orderliness, and cautiousness. Menza *et al*[20] reported that patients with Parkinson's disease were less "novelty seeking" than controls. Low novelty seeking patients were described as being reflective, rigid, stoic, slow tempered, frugal, orderly, and persistent. Some of these are obsessive traits.

In summary, our data support the involvement of basal ganglia in obsessive-compulsive symptomatology.

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