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Binge Eating in Parkinson Disease: Prevalence, Correlates, and the Contribution of Deep Brain Stimulation

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Abstract

Of 96 Parkinson's disease (PD) patients at the University of Florida Movement Disorders Center, one (1%) met diagnostic criteria for binge eating disorder (BED). Eight (8.3%) exhibited subthreshold BED. Psychometric criteria classified problem gambling in 17.8%, hoarding in 8.3%, buying in 11.5%, hypersexuality in 1.0%, and mania in 1.0% of patients. More overeaters met psychometric criteria for at least one additional impulse control disorder (67% vs. 29%). No more overeaters than non-overeaters were taking a dopamine agonist (44% vs. 41%). More overeaters had a history of subthalamic DBS (44% vs. 14%). History of DBS was the only independent predictor of overeating.

Keywords

Parkinson's disease; binge eating; impulse control disorders

INTRODUCTION

Parkinson's disease (PD) is a neurodegenerative disorder characterized by the prominent loss of dopamine neurons in the substantia nigra pars compacta. While first recognized as a purely motor disorder featuring resting tremor, rigidity, bradykinesia and postural instability, it is increasingly being conceptualized as a neuropsychiatric disorder due to the high prevalence of non-motor symptoms.{1} Psychiatric features include depression, apathy, anxiety, psychosis and sleep disturbance.

Recent studies have identified a subpopulation of PD patients who exhibit impulsive and compulsive behaviors, such as pathological gambling, hypersexuality, compulsive shopping, punding and binge eating.{2} Many consider ICDs to result purely from iatrogenic factors, and symptoms often subside when dopaminergic medications are reduced or replaced.{3} However, several patient factors have been variably linked to ICD manifestation, including male sex, younger age at PD onset, personal or family history of substance abuse or other psychiatric disorders, and a personality style characterized by impulsiveness.{3,4} True prevalence rates for ICDs in PD are not well established.{4} However, preliminary evidence supports that ICDs are more common in PD than in the general population or in healthy control subjects. Prevalence estimates range from 0.4 to 10%, depending on the specific ICD.{5} While several authors have described the phenomenon of binge eating amongst PD patients, no reliable point prevalence estimates have been reported.{4} With regard to the

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general population, a recently-completed, multinational study identified binge eating disorder in 1.12% of over 20,000 European participants.{6}

The American Psychiatric Association defines binge eating disorder (BED) as eating an amount of food that is definitely larger than most people would eat during the same period of time under similar circumstances coupled with a perceived lack of control over one's eating (Criterion A). Patients with BED must exhibit marked distress regarding binge eating (Criterion C) and 3 out of 5 associated features (Criterion B). Binge eating episodes must occur at least twice weekly for a six month period (Criterion D), and individuals must not engage in inappropriate compensatory behaviors such as purging or fasting (Criterion E). Many researchers have recently supported the consideration of subthreshold eating disorders in the case of individuals who fail to meet all of the above DSM-IV criteria.{6,7} The purpose of the present prospective study was to determine the prevalence of and factors associated with BED and subthreshold binge eating in Parkinson's disease (PD).

METHODS

Participants

One hundred consecutive PD patients were approached during regularly scheduled follow-up visits at the University of Florida Movement Disorders Clinic, from September to December 2008. All patients carried a current diagnosis of idiopathic PD based on United Kingdom Parkinson's Disease Society Brain Bank Diagnostic Criteria for Parkinson's Disease.{8} We excluded all patients with uncontrolled hypo/hyperthyroidism, brittle diabetes mellitus or significant medical issues that could affect normal dietary habits within the last 60 days prior to the survey.

Procedures

Demographic and disease characteristics were obtained directly from patients and their caregivers. Motor severity was assessed by clinicians specializing in movement disorders using the Unified Parkinson Disease Rating Scale (UPDRS-III). All patients were administered quality of life, cognitive and behavioral assessments, including the Parkinson Disease Questionnaire (PDQ-39), the Beck Depression Inventory-II (BDI-II), the State-Trait Anxiety Inventory (STAI), the Apathy Scale (AS) and the Mini-Mental State Examination (MMSE).{9–12} Next, all patients completed a battery of tests assessing impulsive and compulsive behaviors, as described below. All procedures were approved by the local Institutional Review Board at the University of Florida.

Measures

Impulsive and compulsive behaviors were assessed with a battery of well-validated self-report questionnaires. The Barratt Impulsiveness Scale (BIS-11) contains 30 Likert-type items, and higher scores indicate greater general impulsivity.{13} The Yale-Brown Obsessive Compulsive Scale (YBOCS) contains 19 items, and only the first 10 Likert-type items contribute to the total score.{14} Scores of 0 to 7 indicate "subclinical" symptoms. The Mania Rating Scale (MRS) contains 11 Likert-type items, and scores above 20 indicate significant symptoms.{15} The South Oak Gambling Scale (SOGS) contains 16 items of varying formats.{16} Total score is determined by the number of "at risk" responses. Scores of 1 to 4 indicate "some problems" with gambling, and scores above 4 indicate "probable pathological" gambling. The Sexual Compulsivity Scale (SCS) contains 10 Likert-type items.{17} Suggested cut-scores for elevated sexual compulsivity are 2.1 for men and 1.7 for women. The Saving Inventory-Revised (SI-R) contains 23 Likert-type items and yields a global score and three subscale scores: Clutter, Acquisition, and Difficulty Discarding.{18} A global score above 40 indicates significant hoarding. The Problematic Internet Use

Questionnaire (PIUQ) contains 18 Likert-type items and yields a global score and three subscale scores: Obsession, Neglect, and Control Disorder.{19} A global score above 42.4 indicates signficant problems. The Valence Compulsive Buying Scale contains 13 Likert-type items.{20} Compulsive buyers tend to score 42.2 or higher.

Eating behaviors were assessed with the Eating Disorder Examination Questionnaire (EDE-Q) and the Eating Disorder Diagnostic Scale (EDDS).{21,22} The EDE-Q is a 36-item self-report measure adapted from the EDE interview that features a 7-point Likert-type scale. The EDDS is a 22-item self-report questionnaire containing both yes/no items and Likert-type scales. It assesses DSM-IV Criteria A-E for binge eating disorder. In the present study, responses on the EDDS were used to identify the presence of BED based on DSM-IV criteria: (A) *yes* to items 5 and 6; (B) *yes* to at least 3 of items 9–13; (C) *yes* to item 14; (D) a score of at least 2 on item 7; and (E) scores of 0 on items 15–18. Patients who met Criteria A and E and reported at least weekly binge eating episodes over the previous 6 months but who failed to meet one or more of the additional Criteria were identified as having subthreshold binge eating disorder.{6,7}

Statistical Analyses

Descriptive statistics were used to characterize the sample. Relationships between variables were initially explored using Pearson correlations. Non-parametric Mann-Whitney U tests were used to compare overeaters and non-overeaters on a variety of demographic, disease, and psychological characteristics. Categorical variables were analyzed using Chi square tests. The independent influences of selected variables on overeating status were examined with hierarchical binary logistic regression.

RESULTS

From 100 consecutive patients approached, 96 patients completed the study. The four patients who declined to participate all cited the number of questionnaires as their reason for refusal. Demographic and disease characteristics of the sample are shown in Table 1. Of these 96 patients, 22 (22.9%) had previously undergone deep brain stimulation surgery in either the pallidum (GPi; 6%) or the subthalamic nucleus (STN; 17%), while 74 were exclusively medically managed. Approximately 42% of patients were taking a dopamine agonist when assessed. On average, patients were not demented or depressed. Only one patient scored below the cut-off for dementia (MMSE<24). Seventeen patients evidenced mild to moderate depression (BDI>14).

According to responses on the EDDS, one patient met full diagnostic criteria for BED (1%). An additional 8 patients (8.3%) exhibited subthreshold binge eating in that they all met Criteria A and E for BED. Five patients did not meet Criterion B, 2 patients did not meet Criterion C, and 1 patient did not meet Criteria B, C, or D. This latter individual reported weekly episodes over the last 6 months.

The severity of disordered eating habits was quantified using EDE-Q global scores. In the entire sample, disordered eating correlated with general impulsiveness (r=.585; p<.001) and mania (r=.366; p<.001). No significant correlations were noted between disordered eating and any of the demographic, disease or psychological variables. Table 1 shows that overeaters (N=9) did not differ from non-overeaters (N=87) on any demographic, motor or mood variables. However, overeaters endorsed greater amounts of overall disordered eating, impulsivity, mania, and clutter behaviors than non-overeaters.

Using psychometric criteria described by the individual scales' developers, the prevalence of impulse control behaviors other than binge eating was determined. Note that DSM-IV

clinical criteria were not used to categorize individuals, as a complete diagnostic interview for these other impulse controls disorders was not part of the present protocol. Six patients (6.25%) were identified as probable pathological gamblers. An additional 11 patients (11.5%) had problematic gambling. Eight patients (8.3%) had compulsive hoarding, 11 patients (11.5%) had compulsive buying, one patient (1.0%) had hypersexuality, and one patient (1.0%) had clinically-significant manic symptoms. No pathologic internet use, anorexia nervosa or bulimia nervosa was found.

Next, we examined whether overeaters were more likely than non-overeaters to demonstrate other impulse control behaviors. Table 2 displays the number of overeaters and non-overeaters who met psychometric criteria in one or more of these domains. As shown, overeaters were more likely than non-overeaters to report mania, hoarding, hypersexuality, and compulsive buying. There was a trend for overeaters to be more likely than non-overeaters to exhibit significant obsessive-compulsive features. Six of the 9 overeaters (67%) met psychometric criteria for *at least one* other impulse control disorder, as compared to 25 out of 87 non-overeaters (29%). This difference was significant ($\chi^2(1)=5.367$; Phi=.236; $p=.02$).

Four of the 9 (44%) overeaters were currently on an agonist, as compared to 41% of controls, and this difference was not significant ($\chi^2(1)=0.03$; Phi=.18; $p=.86$). Four out of 9 (44%) overeaters had undergone STN DBS surgery, and 0 had undergone GPi DBS. Twelve (14%) and 6 (7%) non-overeaters had undergone DBS in the STN and GPi, respectively. The association between overeating and DBS history (STN, GPi or no DBS) was at trend ($\chi^2(2)=5.82$; Cramer's V=.246; $p=.055$).

In order to determine the relative contributions of demographic, disease and iatrogenic variables to overeating, a hierarchical logistic regression was conducted in which the dependent variable was eating status (overeater or non-overeater). Based on previous research, the first block comprised iatrogenic variables (DBS, DA agonist therapy, levodopa equivalent daily dose). The second block comprised disease characteristics (age of PD onset, UPDRS-III total score), and the third block comprised demographic characteristics (age, sex, education). Results are displayed in Table 3. While the model did not reach significance at any of the three steps, DBS emerged as the only independent predictor of being an overeater at all three steps ($p=.023$ after Step 1; $p=.021$ after Step 2; and $p=.019$ after Step 3).

DISCUSSION

Our study is cross-sectional, with all of its inherent weaknesses. Nonetheless, the present report describes results from the first prospective study on the point prevalence of binge eating disorder in PD patients in the United States. It provides further support for a relationship between binge eating and other behaviors in the impulsive-compulsive spectrum. Results also suggest that future research should explicitly examine the contribution of deep brain stimulation (DBS) in the subthalamic nucleus (STN) to binge eating behaviors.

We identified the presence of BED in only 1% of our unselected sample of 96 consecutive PD patients, which is comparable to the 1.12% prevalence estimate reported in a recent multinational study. {6} In the future, a larger sample size should be used to confirm this prevalence value. The current estimate of BED prevalence is lower than those reported for pathological gambling and hypersexuality and higher than that reported for compulsive buying in PD.{2} Despite the lack of higher prevalence of BED in this sample of PD patients, we identified subclinical binge eating disorder in 8% of the sample, which is

similar to or higher than rates of other ICDs.{2} Using psychometric criteria, we also identified relatively high levels of problem gambling (11.5%), compulsive buying (11.5%), and hoarding (8.3%) within our sample.

We found substantial support for the co-occurrence of ICD behaviors amongst individuals with PD. Binge eating severity correlated with measures of impulsivity and mania. More overeaters met psychometric criteria for at least one other ICD, as compared to non-overeaters. This overlap is consistent with the current view of a common pathophysiological mechanism underlying these behaviors in PD involving mesocorticolimbic sensitization. Recent neurobiological experiments shave demonstrated enhanced dopaminergic activity within the ventral striatum of PD patients displaying pathological gambling or dopamine dysregulation syndrome (DDS).{26}

Previous studies have variably identified male sex, younger age at PD onset, and depression as risk factors for ICDs.{2} In our sample, overeaters and non-overeaters did not differ on any of these variables. However, most previous studies focused on pathological gambling. Many have failed to identify these variables as independent risk factors when using multivariate analyses.{4,5} No study has yet examined the contributions of these variables exclusively to binge eating in PD. Of note, a recent mail-survey study involving 312 PD patients similarly failed to identify male sex, mean daily levodopa dose, or age of PD onset as statistically significant predictors of ICD behaviors.{27} These results suggest that additional risk factors for these symptoms must be explored.

Surprisingly, we did not find any association between the use of dopamine agonists and the presence of binge eating. Multiple studies have implicated dopamine agonists acting on D3 receptors in the pathophysiology of ICDs and repetitive behavior in PD.{4,5,27,28} However, confounding variables such as differing prescribing practices may have contributed to initial findings.{29} A recent meta-analysis failed to demonstrate a significant relationship between ICDs and individual dopamine agonists.{30} A significant percentage of our cohort underwent DBS surgery, which could have altered the patients' medication regimen post-surgery.

This study identified a history of STN DBS surgery as the only significant predictor of binge eating in a non-selected sample of consecutive PD patients. Further, there was a trend for an association between STN DBS and being an overeater. However, because the present study did not systematically measure and compare ICD symptomatology before and after surgery, it cannot be concluded that DBS *induced* binge eating in these patients. Identification of such a relationship requires future prospective studies with matched controls. None of the four overeaters with a history of STN DBS carried a psychiatric diagnosis prior to surgery. However, it is unknown whether they demonstrated subclinical symptoms. Of note, two of these four overeaters described an increase in their desire for sweets (i.e., candies and ice cream) subsequent to DBS.

Reports of the effects of DBS on impulsive and compulsive behaviors have been somewhat conflicting and have included descriptions of worsened, improved and newly-developed ICDs following surgery.{31–34} All four overeaters with a history of DBS in the present study were implanted in the STN, and it has been suggested that STN DBS results in more non-motor complications than DBS in other targets.{35,36} Further controlled studies are needed to determine the specificity of our findings to DBS in the STN. Interestingly, recent experimental evidence has shown that high frequency stimulation of the STN modulates neurotransmission in limbic regions such as the nucleus accumbens shell, which has been implicated in the pathophysiology of ICDs.{37}

Weight gain following DBS has been documented by numerous groups.{38} Bilateral and unilateral STN stimulation have been associated with weight gains of approximately 10 kg and 4 kg after one year, respectively.{38} Post-surgical weight gain has been attributed to medication reduction, improved ability to eat, and/or decreased basal energy expenditure. {39} Despite reports of uncontrolled appetitive behaviors following DBS, the potential contribution of increased frequency of binge eating episodes has not yet been systematically evaluated.

The relatively high prevalence of clinical and subclinical symptomatology reported here suggests that physicians should be aware of binge eating in their PD patients, especially considering the potential adverse consequences of prolonged occurrences. The relationship between binge eating and weight gain following DBS surgery should also be elucidated, as it is important for physicians and patients to be cognizant of potential surgical side effects during treatment planning.

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Table 1

Demographics, disease characteristics, and group comparisons

	Overeaters (N=9) Mean (SD)	Non-overeaters (N=87) Mean (SD)	Mann-Whitney U or χ^2
Demographic			
Age	68.3 (4.5)	65.8 (10.3)	318
Sex	6 M/3 F	57 M/30 F	.005
Education	14.4 (3.0)	14.7 (2.8)	324
Disease/Health			
Age at PD onset	57.97 (7.8)	55.8 (12.8)	364.5
Months with Symptoms	124.3 (56.9)	119.3 (109.1)	322.5
UPDRS-III	28.7 (7.0)	31.1 (11.1)	331.5
LED	563.1 (250.9)	680.8 (490.4)	274.5
Weight	176.8 (36.3)	179.7 (44.6)	386
BMI	29.2 (7.1)	29.3 (7.9)	391
Neuropsychological			
MMSE	29.3 (0.7)	28.8 (1.5)	306
BDI-II	7.9 (9.9)	8.1 (5.8)	326.5
STAI – State	36.0 (16.1)	38.0 (10.6)	318
STAI – Trait	38.1 (17.0)	36.0 (9.9)	390
AS	14.8 (9.2)	13.6 (12.1)	352
Study Measures			
EDE-Q Global	1.7 (1.2)	0.5 (0.8)	102**
EDE-Q Restraint	1.5 (1.8)	0.4 (0.8)	189.5**
EDE-Q Eating Concern	3.2 (2.2)	0.7 (2.7)	84**
EDE-Q Shape Concern	0.6 (1.2)	0.1 (0.4)	262**
EDE-Q Weight Concern	1.4 (1.5)	0.8 (1.2)	239.5*
BIS	19.4 (10.1)	4.6 (6.8)	68**
YBOCS	4.8 (9.5)	0.7 (2.9)	329.5
YMRS	4.7 (6.6)	1.4 (3.0)	204**
SOGS	1.4 (3.1)	0.7 (2.1)	364.5
SCS	1.2 (0.4)	1.1 (0.2)	365.5
SI-R	26.6 (16.3)	17.5 (11.7)	256+
SI-R Clutter Subscale	8.8 (6.1)	4.7 (5.0)	210.5*
Compulsive Buying	36.3 (20.0)	21.2 (12.2)	201*
PIUQ	20.7 (5.5)	20.9 (6.9)	376

* $p<.05$.

** $p<.01$.

+ $p<.1$.

Table 2

Proportions of individuals meeting criteria for other impulsive control psychopathology

Classification	Overeaters (N=9)	Non-overeaters (N=87)	Pearson chi square	*p*	Phi
Mania	1 (11.1%)	0 (0%)	9.77	.002	.32
Hoarding	3 (33.3%)	5 (5.7%)	8.13	.004	.29
Problem Gambling	2 (22.2%)	15 (17.2%)	0.40	.82	.07
Compulsive Buying	4 (44.4%)	7 (8.0%)	10.65	.001	.33
Obsessive-Compulsive	2 (22.2%)	5 (5.7%)	3.28	.07	.19
Sexual Compulsivity	1 (11.1%)	0 (0%)	9.77	.002	.32
Pathological Internet Use	0 (0%)	0 (0%)			
At least one of above	6 (66.7%)	25 (28.7%)	5.37	.02	.236

Table 3

Hierarchical logistic regression results

	B	*SE B*	exp *b*	*p*
Step 1				
Constant	0.019	1.202	1.019	.988
DBS	−2.109	0.927	0.121	.023*
DA agonist	−0.130	0.841	0.878	.878
LEDD	−0.002	0.001	0.998	.192
Step 2				
Constant	−1.908	2.706	0.148	.481
DBS	−2.679	1.163	0.069	.021*
DA agonist	0.052	0.876	1.053	.953
LEDD	−0.002	0.001	0.998	.182
UPDRS-III	−0.021	0.048	0.980	.668
Age of PD Onset	0.050	0.054	1.052	.354
Step 3				
Constant	−3.722	4.448	0.024	.024
DBS	−2.688	1.142	0.068	.019*
DA agonist	0.234	0.908	1.264	.796
LEDD	−0.002	0.001	0.998	.147
UPDRS-III	−0.029	0.050	0.971	.562
Age of PD Onset	−0.005	0.051	0.995	.917
Age	0.080	0.070	1.083	.257
Sex	0.046	0.958	1.047	.961
Education	−0.002	0.173	0.998	.989

R^2 = .069 (Cox & Snell), .155 (Nagelkerke). Model $\chi^2(3)=5.777$, $p=.123$ for Step 1.

ΔR^2 = .011 (Cox & Snell), .025 (Nagelkerke). Model $\chi^2(5)=10.463$, $p=.234$ for Step 2.

ΔR^2 = .012 (Cox & Snell), .026 (Nagelkerke). Model $\chi^2(8)=10.979$, $p=.203$ for Step 3.

* $p<.05$.