Cognitive-Behavioral Therapy for Pathological Gambling in Parkinson’s Disease: A Pilot Controlled Study

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Abstract

Objectives: The main objective of this study was to compare the clinical characteristics and differences in response to treatment of two groups of pathological gamblers: with comorbid Parkinson’s disease (PG + PD) and without (PG – PD).

Methods: Clinical and psychopathological profiles and response to cognitive-behavioral treatment were assessed in 15 PG + PD and 45 PG – PD individuals consulting a specialized hospital Unit.

Results: Statistically significant differences were observed between the two groups on a series of clinical variables. PG + PD patients were older and presented later onset of problematic gambling behaviors, lower alcohol consumption and higher bingo playing than PG – PD patients. No significant differences were noted in psychopathology except for lower measures of hostility in the PG + PD group. No statistical differences were detected between groups in terms of response to treatment.

Conclusion: These results may provide guidance for obtaining accurate diagnostic information in pathological gamblers by properly identifying patients with specific needs that may be targeted with treatment.

Introduction

Pathological gambling (PG), included as an impulse control disorder not elsewhere classified in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision [1], is characterized by a failure to resist the impulse, drive or temptation to perform an act that is harmful to the person or to others. Impulse control disorders share biopsychological vulnerabilities and clinical and psychopathological characteristics, expressed in the form of neuropsychological deficits [2, 3], personality traits, namely impulsivity or risk-taking [4], and pathological impairment [5]. In individuals with PG, higher impulsivity has been associated with specific subgroups of patients, severity of the disorder, poorer psy-
Impulse Control Disorders and Parkinson’s Disease

Impulse control disorders are relatively common in Parkinson’s disease (PD), with an estimated prevalence of 9.3% compared to 1.6% in the general population [11]. Furthermore, PG has been reported as a complication in the treatment of PD [12–14]. In fact, PG is one of several disturbances recognized to arise de novo in the context of PD treatment with dopaminergic medications [15, 16]. Other behaviors thought to be induced by this type of pharmacological treatment include the addictive use of dopamine replacement therapy, stereotyped behaviors, known as punding, and other impulse control disorders, such as hypersexuality, compulsive buying and compulsive eating. Although dopamine replacement therapy is thought to play a key role in the reward and incentive system, the fact that PG emerges in only a subset of individuals treated with dopaminergic drugs suggests an interaction with individual vulnerabilities. Younger age at PD onset [17, 18], personal and family history of alcohol abuse [19, 20] and impulsive and novelty-seeking personality traits [14] have been proposed as risk factors for the development of PG in PD. Although the pathophysiological mechanism whereby dopaminergic drugs may trigger PG in PD is not known, some studies are suggesting that this might be due to hyperfunction in specific brain areas, such as subthalamic and limbic region [21, 22], but also to greater release of dopamine in the ventral striatum [23].

Cognitive-Behavioral Therapy in PG and PD

Traditionally, PD has not only been associated with motor symptoms and cognitive deficits, but also with affective symptoms and behavioral deterioration along with the progression of the disease [24]. Although cognitive-behavioral therapy (CBT) has been demonstrated to be effective for treating PG [6, 25, 26] and for the management of depressive symptomatology in PD [27, 28], there is a lack of studies that focus on the use of CBT to treat PG in patients with PD. Testing nonpharmacological treatment for the management of impulse control disorders in PD is particularly important given that many PD patients with PG are reluctant or unable to decrease the dosage of their dopaminergic medication [29]. Thus, we consider that comparing treatment responses of PD and non-PD patients with PG could inform best practices for the treatment of these complex disorders.

Aims of the Study

The specific goals of this research were threefold: (a) to assess clinical and psychopathological characteristics in individuals with and without PD who are pathological gamblers (PG + PD versus PG – PD); (b) to characterize differences in therapy response between PG – PD and PG + PD groups; (c) to assess the main predictors of developing PG + PD taking into account severity of the disorder, sociodemographical and psychometrical characteristics.

Hypotheses

Based on the few reports in the literature where clinical features of PD patients with PG were comparable to those described in clinical samples of PG [30], we hypothesized that the therapy response in both conditions would be similar.

Methods

Participants

Subjects were recruited for the study between September 2004 and February 2009. The initial sample included 1,182 PG patients consecutively admitted to the Pathological Gambling Unit at the University Hospital of Bellvitge. Individuals were excluded from the analyses if they had missing values in any of the relevant items (n = 202). Of the 980 remaining patients, 15 PG patients (1.47% of the total) presented PD and were included in the present study. For all of the PD patients, PD was a premorbid medical condition (development of PG after diagnosis of PD and initiation of dopamine replacement therapy). The selected patients had been referred to our specialized unit by neurologists and primary care physicians for treatment of PG. While in therapy, PD patients remained under the care of their physicians. Individuals were excluded from the analyses if they had missing values for any diagnostic items. To increase the statistical power, a pairwise matching comparison procedure, was used to form a comparison sample of 45 patients diagnosed with PG. Each participant was paired with a randomly selected patient with the same PG diagnosis (in our unit we see patients with other impulse control disorders and several forms of behavioral addictions), sex and type of therapy (specifically individual CBT). Randomization was performed by computer. All participants were diagnosed using DSM-IV criteria obtained through a semi-structured clinical interview (SCID-I), conducted by experienced psychologists and psychiatrists. The mean age of the whole sample was 46.2 years (SD = 16.1). The mean age at onset of the PD was 41.0 years (SD = 15.9), and the mean duration of the disorder was 5.0 years (SD = 3.9). All PG + PD patients were taking at least one dopamine agonist; of these, 14 patients started gambling after the onset of PD and the initia-
tion of pharmacotherapy. The mean age at onset of pharmacotherapy for PD was 55.6 years (SD = 10.3).

This study was carried out in accordance with the latest version of the Declaration of Helsinki. The Ethics Committee of the University Hospital of Bellvitge approved the study, and informed consent was obtained from all participants.

Assessment
We developed a comprehensive battery of assessments to identify PG symptoms, general psychopathology, and personality. The battery included the South Oaks Gambling Screen (SOGS) [31], a 20-item diagnostic questionnaire that effectively discriminates between probable pathological gamblers, problematic gamblers, and non-problematic gamblers. A Spanish validation of this questionnaire [32] showed high reliability and validity: its test-retest reliability is 0.98 and internal consistency 0.94 (Cronbach’s alpha). And its convergent validity with regard to DSM-III-R criteria for reliability is 0.98 and internal consistency 0.94 (Cronbach’s estimated as r = 0.77 for the general population and 0.75 for a gambling treatment group. Convergent validity was estimated using a measure of internal consistency was explained during these initial sessions, and therapeutic materials additional demographical information during two structured face-to-face interviews. As well as a comprehensive clinical and psychological evaluation (including somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychotismic. (3) The Global Severity Index, which is the participant’s mean score (using all the 90 items), is a widely used global index of distress. The Spanish validation of this scale shows good psychometric properties, with internal consistencies ranging from 0.81 to 0.90. Test-retest reliability ranged from 0.78 to 0.90. We obtained additional demographic information, including age, marital status, education, occupation, living arrangements and clinical relevant variables (namely age at onset, problem gambling, frequency, mean/bet size, and smoking status) via a semi-structured interview.

Procedure and Design
The patients filled in all the study questionnaires at the start of treatment at our Unit. Three experienced psychologists and psychiatrists (neuroscience master and doctorate level, with more than 15 years working in PG and specially trained in liaison psychology for treating chronic illnesses, completed the intake evaluation during two structured face-to-face interviews. As well as a comprehensive clinical and psychological evaluation (including the instruments mentioned previously) additional demographical data were also collected. The therapeutic approach and rationale was explained during these initial sessions, and therapeutic materials were provided. Patients underwent a CBT intervention based on Sharpe and Tarrier’s [41] integrative model, consisting of 16 weekly outpatient sessions (45 min each), whose structure and format was adapted to individual patient’s needs. The main objective of the treatment was to teach patients to use CBT strategies so as to achieve complete abstinence from problem gambling. As described previously [6], the topics addressed included psychoeducation on the disorder, stimulus control, response prevention, cognitive restructuring, reinforcement and self-reinforcement, social skill training and relapse prevention techniques. Additionally, self-monitoring records about adherence, relapses, and achievement of intersession tasks were systematically collected during every treatment session. This treatment program and accompanying program materials have already been manualized and published in Spanish [42]. To fit the therapeutic needs of PD patients, we adapted our initial therapy model. Therefore, several topics were more emphasized in the PG + PD group than in the PG group. These included: psychoeducation about specific conditions, self-instructions and planning of alternative gratifying activities (e.g. interaction between dopaminergic drug therapy and gambling behavior, additional impulsive behaviors to take into account, how to cope with somatic chronic disease and how to deal with free time; see table 1).

Statistical Analysis
Statistical analysis was carried out with SPSS 15.0.1 for Windows. We assessed the number of treatment sessions; dropout and relapse during the 16-week intervention and total SOGS scores.

To establish the rate of dropout and relapse, we only considered the data from patients who completed treatment. Treatment dropout was defined as no therapy attendance for more than three sessions. Relapses were defined as any episode of gambling (commercial and non-commercial, involving a money bet) during the 16 weeks of treatment. Although both variables were not mutually exclusive, for the statistical analysis they have been considered separately. What was defined as poor performance was weak compliance with the treatment regimens, namely bringing the self-monitoring diaries to the weekly sessions and regularly attending the group meetings.

In the first step, clinical profiles (severity of PG measures and global psychopathology) were compared through Mann-Whitney tests for patients diagnosed with PG and those with PG and PD. In addition, the specific predictive accuracy of PD on clinical outcomes was measured with the change in R² coefficient obtained in hierarchical multiple regressions, entering the patient’s age in the first step and the presence of PD in the second step. In the second step, the risk (cumulative incidence) of therapeutic outcomes (presence of relapses and dropouts) was obtained and compared for patients diagnosed with PG and PG with PD through logistic regressions adjusted for age. The goodness-of-fit was valued with Hosmer-Lemeshow’s test, and the discriminative accuracy with the area under the ROC curve (AUC).

Finally, survival analysis techniques were used to model the rate of relapses and dropouts during the 16 sessions of treatment (over 4 months). Survival functions adjusted for sex, age and severity of PG (SOGS total score) were obtained and compared for patients with PG alone or PG with PD. Next, a multiple Cox’s regression valued the specific contribution of patient’s age and sex, severity of PG (SOGS score) and the presence of PD to the relapse and dropout rates. Global predictive accuracy was measured with the R² with Atkinson estimation [43].
Results

Clinical and Sociodemographic Characteristics of the Sample
The between-group comparison (table 2) suggested an older age and older age at onset ($p < 0.0005$) in the PG + PD group, although duration of PG was similar in both groups ($p = 0.337$). No significant differences were found in gender distribution ($p = 0.999$), educational level ($p = 0.076$) or alcohol and drug abuse ($p = 0.680$ and $p = 0.173$). The PG – PD group had higher percentages of smokers ($p < 0.0005$) and employment ($p = 0.001$) and a lower rate of divorce ($p = 0.014$) than the PG + PD group. Differences were also noted in the type of gambling problem, with significantly more bingo playing in the PG + PD group ($p = 0.042$).
Psychopathological and Clinical Indices

Table 3 shows the results of the first step of clinical profile analysis (severity of PG measures and global psychopathology). No statistically significant differences were found between PG – PD and PG + PD in severity of the disorder (SOGS p = 0.111; DSM-IV p = 0.299) and psychopathologic profiles (SCL-90-R score, SOGS questionnaire, and Stinchfield’s questionnaire), with the exception of higher scores on the Hostility Scale (p = 0.005) and Positive Symptom Total (number of self-reported symptoms, p = 0.010) for the PG – PD group.

Hierarchical multiple regressions (entering the patient’s age in the first step and the presence of PD in the second step) revealed no significant associations between presence of PD, the rate of global psychopathology and severity of PG.

Table 4 shows that the risk of relapses was 24.4% for the PG – PD group and 41.7% for the PG + PD group, and the OR adjusted for patient’s age was not statistically significant (OR = 5.26, p = 0.088). The risk of dropout was statistically equal for both diagnoses (28.9% for PG – PD and 33.3% for PG + PD, OR = 2.48, p = 0.305), as was the probability of low compliance with therapy (35.6% for PG – PD versus 33.3% for PG + PD, OR = 0.825, p = 0.811). Discriminative accuracy was moderate to low (AUC values between 0.57 and 0.66).

Survival Analyses for the Rate of Relapses and Dropouts

Figure 1 shows that the weekly rate of relapses and drop-out during the 16 sessions of treatment was higher for the PG + PD group. At the end of the first month, 25%
### Table 3. Comparison between PG – PD and PG + PD of psychopathological and clinical outcomes at baseline

<table>
<thead>
<tr>
<th></th>
<th>PG – PD (n = 45)</th>
<th>PG + PD (n = 15)</th>
<th>M-W p</th>
<th>Hierarchical regressions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
<td>Results obtained in the second step</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B</td>
</tr>
<tr>
<td>SOGS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total score</td>
<td>10.24 ± 2.9</td>
<td>8.73 ± 3.5</td>
<td>0.111</td>
<td>−0.457</td>
</tr>
<tr>
<td>DSM-IV</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Total score</td>
<td>7.18 ± 1.9</td>
<td>6.54 ± 2.0</td>
<td>0.299</td>
<td>−0.029</td>
</tr>
<tr>
<td>SCL</td>
<td></td>
<td></td>
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<tr>
<td>Somatization</td>
<td></td>
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<tr>
<td></td>
<td>0.92 ± 0.7</td>
<td>0.98 ± 0.6</td>
<td>0.687</td>
<td>−0.067</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td></td>
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<tr>
<td></td>
<td>1.05 ± 0.7</td>
<td>0.74 ± 0.5</td>
<td>0.214</td>
<td>−0.27</td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.89 ± 0.7</td>
<td>0.69 ± 0.6</td>
<td>0.517</td>
<td>−0.08</td>
</tr>
<tr>
<td>Depressive</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>1.37 ± 1.0</td>
<td>1.10 ± 0.7</td>
<td>0.517</td>
<td>−0.21</td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>0.88 ± 0.7</td>
<td>0.94 ± 0.6</td>
<td>0.724</td>
<td>0.12</td>
</tr>
<tr>
<td>Hostility</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>0.81 ± 0.8</td>
<td>0.18 ± 0.2</td>
<td>0.005</td>
<td>−0.45</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.35 ± 0.6</td>
<td>0.36 ± 0.5</td>
<td>0.779</td>
<td>0.048</td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>0.68 ± 0.7</td>
<td>0.82 ± 0.7</td>
<td>0.670</td>
<td>0.31</td>
</tr>
<tr>
<td>Psychotic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.71 ± 0.6</td>
<td>0.59 ± 0.6</td>
<td>0.652</td>
<td>−0.06</td>
</tr>
<tr>
<td>GSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.93 ± 0.6</td>
<td>0.75 ± 0.5</td>
<td>0.685</td>
<td>−0.15</td>
</tr>
<tr>
<td>PSDI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.86 ± 0.6</td>
<td>1.34 ± 0.8</td>
<td>0.053</td>
<td>−0.50</td>
</tr>
<tr>
<td>PST</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>42.2 ± 21.2</td>
<td>18.2 ± 23.8</td>
<td>0.010</td>
<td>−24.2</td>
</tr>
</tbody>
</table>

M-W = Mann-Whitney test; ΔR² = change in R² coefficient; GSI = Global Severity Index; PSDI = Positive Symptom Distress Index; PST = Positive Symptom Total. Values for PG – PD and PG + PD are expressed as mean ± SD.

### Table 4. Predictive accuracy of PD on therapy outcomes

<table>
<thead>
<tr>
<th></th>
<th>PG – PD (n = 45)</th>
<th>PG + PD (n = 15)</th>
<th>Logistic regression adjusted for age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>OR</td>
</tr>
<tr>
<td>Relapses</td>
<td>24.4%</td>
<td>41.7%</td>
<td>5.26</td>
</tr>
<tr>
<td>Dropout</td>
<td>28.9%</td>
<td>33.3%</td>
<td>2.48</td>
</tr>
<tr>
<td>Poor compliance a</td>
<td>35.6%</td>
<td>33.3%</td>
<td>0.825</td>
</tr>
</tbody>
</table>

H-L = Hosmer-Lemeshow’s test for goodness of fit.

a To be considered with caution due the lack of goodness of fit for this model.

### Table 5. Predictive value of patient’s profile on survival time to therapy outcomes

<table>
<thead>
<tr>
<th></th>
<th>Relapses (R² = 6.4%)</th>
<th>Dropout (R² = 6.5%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>p value</td>
</tr>
<tr>
<td>Sex</td>
<td>−0.055</td>
<td>0.944</td>
</tr>
<tr>
<td>Age</td>
<td>−0.038</td>
<td>0.159</td>
</tr>
<tr>
<td>Severity of PG (SOGS)</td>
<td>−0.029</td>
<td>0.747</td>
</tr>
<tr>
<td>Presence of PD</td>
<td>1.45</td>
<td>0.092</td>
</tr>
</tbody>
</table>

Results obtained through Cox regression models.
of PG + PD patients had reported relapses and 29% had dropped out of therapy, compared with corresponding figures of 11% and 9% in the PG – PD group.

Table 5 shows the Cox’s regression results were not statistically different for the presence of PD and the rate of relapses (OR = 4.25, p = 0.092), entering the patient’s sex, age and severity of PG (SOGS total score) simultaneously. No significant differences were found for the presence of PD and the rate of dropout (OR = 2.38, p = 0.272). Global predictive accuracy was low for both models (R² around 6.5%).

Discussion

The present study aimed to explore differences in clinical and psychopathological features and response to therapy between a group of individuals with PG + PD and a group with PG – PD. The therapeutic approach in PD has often privileged the treatment of motor aspects of disease, whereas non-motor aspects have received little attention from researchers. Although the literature stresses the contribution of non-pharmacological approaches such as CBT or psychosocial support in PD [17, 18, 44, 45], to our knowledge this is the first study that analyzed the effectiveness of CBT in PD with PG.

Clinical and Psychopathological Features in PG + PD and PG – PD

As reported above, most pathological gamblers in our sample were male; the main gambling problem was slot machines and bingo, and the mean problem duration was 5 years [46–49]. Compared to PG – PD, PG + PD patients showed lower rates of smoking and employment. The lower rate of employment is probably attributable to the older age and therefore a higher likelihood of retirement in the PG + PD group, while the differential pattern of substance use corroborates previous reports of a negative relationship between tobacco smoking and the development of PD [50, 51]. Moreover, a study of 498 subjects diagnosed with PG identified a negative relationship between age and the probability of tobacco use (younger, more likely to smoke) [52]. As such, the majority of studies with pathological gamblers observe a positive relationship between consumption of nicotine, alcohol and other substances and adolescence youth [53, 54].

Consistent with the view that the use of dopaminergic medications is associated with PG onset [15–18], gambling behavior in our sample appeared after the onset of treatment with dopaminergic treatment for PD in 15 patients. Our finding corroborates other reports that suggest that PG is a possible consequence of pharmacological treatment [55–57] and could explain the later age at onset of PG in this group. Normally, male pathological gamblers begin to gamble during their youth [58]. The first symptoms, and in fact the complete clinical profile, usually emerge earlier in life [59].

Interestingly, we did not find statistically significant between-group differences on indices of global psychopathology and severity of PG. The lack of difference could be linked to differences in the mean age of the sample and the age at onset of PG. However, the results of research in this area are contradictory. Some studies associate the later age at onset with higher psychopathology [59–62], but other authors have observed more medical and psychiatric problems in early-onset PG [59–61]. In our sample, the age factor may have been mitigated by the presence of pharmacological use as well as by variables related to the illness.

Therapy Response in PG + PD and PG – PD

Finally, accordingly to what we predicted in our study hypotheses, we observed that the presence of PD in PG was not related to treatment response. No significant differences were observed in any of the treatment variables explored. Beyond the statistical results and from a clinical point of view, the obtained response to treatment in both groups is in keeping with previous studies in PG, in which dropout rates between 30–50% are described [6, 63, 64] after standard CBT programs (6–16 weekly sessions plus follow-up, averaging 17 months after end of the treatment) [65]. Regarding relapses, some authors [65, 66] consider that a great number of patients with PG relapse at least once while attempting to quit gambling. As in other addictions, relapses in PG during treatment are frequent, with reported rates between 40 and 70% throughout the treatment follow-up period [67, 68]. Thus, our dropout and relapse rates closely match the percentages described in other studies, and lend support to the use of a CBT-based approach for treating PG in patients with PD, albeit with the expectation of a modest treatment response. Of note, pathological gamblers with PD may be very heterogeneous, ranging from cases that require only an adjustment in dopaminergic pharmacotherapy to resolve their gambling problem to cases that present associated factors of vulnerability such as impulsiveness, low tolerance of stress or low adaptation to PD and finally to cases who suffer comorbid psychiatric disorders (alcoholism, personality disorders, etc.) [69]. Several studies have found that the existence of comorbid disorders compli-
icates the evolution and severity of gambling problems [7, 8]. Most studies support the need to design treatment programs that include appropriate treatment strategies for comorbid disorders in order to ensure good treatment response [7, 69]. To maximize its effectiveness in PD patients, CBT should also be tailored to their particular needs (cognitive deficits such as difficulties with memory, attention and language and other comorbid psychiatric conditions) to decrease the risk of relapse and dropout during treatment.

Limitations

These results should be considered within the context of several limitations. First, the study’s retrospective nature and the self-report data collection procedures may limit the validity of the findings, which are subject to the unreliability of individual recall and potential memory bias, especially in PG + PD patients. Second, the sample size in the present study was very small and could therefore have influenced the results. Third, the assessment procedures used did not allow for in-depth evaluation of comorbid disorders. Fourth, while the definition of relapse is heterogeneous in the addiction literature and has been considered in several different ways [68], the criteria of relapse used in our study are perhaps too strict, not allowing differentiation between patients who presented an isolated episode of relapse versus those who never really stopped gambling. Finally, although we used indirect measures of adherence and compliance (namely frequency of attending therapy sessions and completing weekly homework, as shown in fig. 1), we did not assess our patients with any specific direct measures (e.g. self-reports).

Contributions

A major strength of this study are the specific characteristics of the selected sample. We assessed and compared clinical and psychopathological profiles as well as response to CBT in a group of PG individuals (with and without associated PD) who were treated consecutively at our specialized unit for PG disorder. In summary, although PG individuals (with and without PD) presented similar symptoms, as far as their gambling disorder was concerned, we found certain PD-specific clinical and psychopathological characteristics but also PD-specific treatment response. Our results support the use of CBT in PG patients, although the program must be adapted to their specific needs.

Acknowledgements

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