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Pathological Gambling Following Subthalamic Nucleus Deep Brain Stimulation in Parkinson's Disease: A Case Report and Literature Review

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Abstract | **Case Report**

***Introduction*:** Deep brain stimulation of the subthalamic nucleus (STN-DBS) is a standard treatment for advanced Parkinson's disease (PD). While effective for motor symptoms, it can induce neuropsychiatric complications, including impulse control disorders (ICDs) like pathological gambling (PG). We report a case of *de novo* PG following STN-DBS. ***Case Presentation*:** A 58-year-old man with a 12-year history of PD underwent bilateral STN-DBS for refractory tremor. He had no prior psychiatric or gambling history. One-month post-surgery, while still on a reduced dose of pramipexole, he developed severe compulsive gambling behavior. ***Management and Outcome*:** The management involved a two-pronged approach: discontinuation of the dopamine agonist and careful adjustment of DBS parameters to favor dorsal contacts. This strategy led to the complete resolution of gambling behavior within months, while the excellent motor benefits from DBS were maintained. ***Discussion*:** This case highlights the complex and paradoxical role of STN-DBS in impulse control. It underscores that stimulation itself, likely via modulation of non-motor (limbic) circuits, can be a key precipitating factor, especially in conjunction with dopaminergic therapy. A multidisciplinary management strategy is essential for successful outcomes. ***Conclusion*:** *De novo* PG is a serious but manageable complication of STN-DBS. Thorough preoperative screening, patient and family education, and vigilant postoperative monitoring are essential to mitigate risks and ensure the therapy's success.

Keywords: Parkinson's Disease, Deep Brain Stimulation, Subthalamic Nucleus, Impulse Control Disorder, Pathological Gambling; Case Report.

1. INTRODUCTION

Deep brain stimulation (DBS) of the subthalamic nucleus (STN) is an established treatment for advanced Parkinson's disease (PD), offering substantial improvement in motor symptoms and quality of life [1]. However, its non-motor effects are increasingly recognized. Impulse control disorders (ICDs), such as pathological gambling (PG), hypersexuality, and compulsive shopping, occur in a significant subset of PD patients, most frequently linked to dopamine agonist therapy [2].

The relationship between STN-DBS and ICDs is complex and seemingly paradoxical. On one hand, by enabling a significant reduction in dopaminergic medication, DBS can lead to the resolution of pre-existing medication-induced ICDs [3]. On the other hand, a growing number of case reports and series describe the emergence of *de novo* ICDs post-operatively, suggesting a direct role of stimulation itself in altering reward and impulse circuitry [4]. This report presents a case of *de novo* PG following bilateral STN-DBS, discusses its management based on a review of the literature, and explores the underlying neurobiological mechanisms.

2. CASE PRESENTATION

A 58-year-old man with a 12-year history of idiopathic PD was referred for DBS evaluation due to severe medication-refractory tremor, motor fluctuations, and disabling dyskinesias. There was no prior personal or family history of psychiatric illness, impulse control disorders, or gambling. Preoperative neuropsychological assessment revealed normal global cognition (Mini-

Mental State Examination score of 28/30) and no evidence of depression or behavioral disorders. His medication regimen included levodopa-carbidopa (total daily levodopa equivalent dose of ~800 mg) and the dopamine agonist pramipexole (3 mg/day).

The patient underwent uneventful bilateral STN-DBS implantation (Figure.1). Postoperative programming was optimized over several sessions, resulting in a dramatic improvement of his tremor, rigidity, and bradykinesia. This motor benefit allowed for a gradual reduction of his dopaminergic medication; the pramipexole dose was halved to 1.5 mg/day in the first month.

Approximately four weeks after the initiation of chronic stimulation, the patient's family noted subtle behavioral changes. He developed an intense, compulsive interest in lottery tickets and began visiting a local casino. This behavior escalated rapidly over the following weeks, leading to significant financial losses. He met the DSM-5 criteria for Gambling Disorder. No other ICDs or manic symptoms were observed.

A comprehensive multidisciplinary assessment was conducted. The working diagnosis was a DBS and dopamine agonist-related PG. Management involved:

1) **Complete tapering and discontinuation of pramipexole.**
2) **Reprogramming of the DBS stimulators:** The active contacts were switched to more dorsal configurations to minimize current spread to the ventral, limbic regions of the STN.
3) **Psychosocial interventions:** The family assumed control of finances, the patient agreed to self-exclusion from gambling venues, and he was referred for cognitive-behavioral therapy.

The outcome was favorable. Within one month of intervention, the gambling urges subsided significantly. By six months, the behavior had resolved completely without any recurrence at the one-year follow-up. The motor benefits from DBS were excellently maintained on levodopa monotherapy alone.

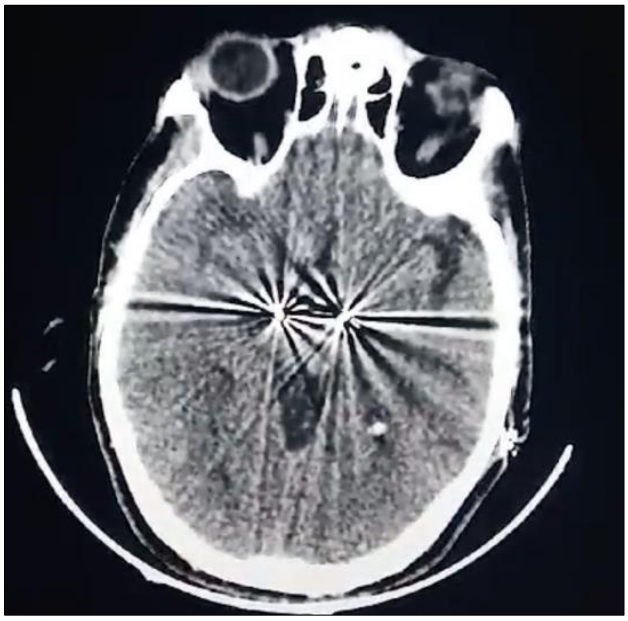

Figure 1: Brain CT of the patient A.A, showing the targeted subthalamic nuclei

3. DISCUSSION

Our case illustrates a serious neuropsychiatric complication of an otherwise highly effective therapy: the emergence of a *de novo* impulse control disorder following STN-DBS. The clear temporal association with the surgery and the resolution upon therapeutic adjustments provide a strong argument for an iatrogenic cause.

Neurobiological Mechanisms and the "Dual Hit" Hypothesis

The pathophysiology of PG in this context likely involves a "dual hit" mechanism. First, the dopaminergic medication, particularly dopamine agonists, exerts its effect by overstimulating mesolimbic D3 receptors in the ventral striatum, enhancing reward-seeking behavior [5]. Second, STN-DBS itself may modulate the neural circuits governing impulse control. The STN is not a purely motor structure; it has associative and limbic territories interconnected with prefrontal cortex and the nucleus accumbens [6]. High-frequency stimulation functionally inhibits the STN. If this inhibition impacts the limbic territory, it could reduce the "braking" signal the STN applies to impulsive actions, as proposed in computational models of decision-making [7]. In our patient, STN-DBS may have induced a state of limbic disinhibition, while the residual dose of dopamine agonist provided the drive for

compulsive behavior, creating a perfect storm for PG to emerge.

This hypothesis is supported by experimental evidence. Rogers *et al*., demonstrated that STN-DBS can transiently increase impulsive decision-making and "loss-chasing" behavior in PD patients performing gambling tasks [8]. Furthermore, the identical case described by Smeding *et al*., where *de novo* PG resolved after agonist withdrawal and DBS adjustment, strongly reinforces this causal link [9].

Controversies and Risk Management

Our observation sits at the heart of a broader debate on the impact of STN-DBS on impulsivity. While it is a well-documented treatment for medication-refractory ICDs by allowing drug reduction [10], it is also clear that it can *unmask* or *induce* new ICDs [4]. This paradox highlights the delicate balance between dopaminergic tone and circuit modulation, influenced by individual patient vulnerability, precise electrode placement, and post-operative management. It underscores that the risk of ICDs does not vanish after surgery but rather shifts from a purely pharmacological to a neuromodulatory domain.

Clinical Implications: A Proactive and Multidisciplinary Approach

The key lesson from this case is the imperative need for a proactive, multidisciplinary strategy throughout the DBS process:

1. **Preoperative Screening:** Must be meticulous, using validated tools like the Questionnaire for Impulsive-Compulsive Disorders in PD (QUIP) to detect subclinical tendencies.
2. **Patient and Family Education:** is non-negotiable. They must be warned about potential behavioral changes and are often the first to detect them.
3. **Postoperative Monitoring:** must be vigilant, especially in the first year. A structured management protocol is essential:
   - **First Step: Pharmacological Adjustment.** Tapering and discontinuing dopamine agonists is the most effective initial step.
   - **Second Step: DBS Reprogramming.** If problems persist, adjusting parameters to use more dorsal contacts and avoid ventral limbic spread is a critical strategy, as successfully employed here.
   - **Third Step: Psychosocial Support.** Financial management, CBT, and support groups are vital adjuncts.

Limitations

The primary limitation of this study is its nature as a single case report. While suggestive of causality, it cannot prove it definitively or establish prevalence. Furthermore, the simultaneous adjustment of medication and stimulation parameters makes it difficult to isolate the precise contribution of each factor to the resolution of the gambling behavior.

4. CONCLUSION

Pathological gambling can emerge as a *de novo* complication following STN-DBS, underscoring the profound impact neuromodulation can have on behavior-regulating circuits. Clinicians must maintain a high index of suspicion for ICDs in the postoperative period. This case demonstrates that a structured, multidisciplinary management strategy involving dopamine agonist withdrawal, careful DBS parameter optimization, and psychosocial support can successfully resolve this debilitating behavior while preserving the crucial motor benefits of DBS. This approach is essential for achieving truly optimal outcomes for patients undergoing this powerful therapy.

Statements and Declarations

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Ethical Approval: Ethical approval for this case report was waived by the local institutional review board.

Patient Consent:

Written informed consent was obtained from the patient for publication of this case report and any accompanying information. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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