CASE REPORT

Right Basal Ganglia Infarct and the Late Manifestation of Hypersexuality in Schizophrenia: A Case Report

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ABSTRACT

While the basal ganglia are primarily recognized for their motor control, they also play a critical part in regulating goal-directed behaviour, social interaction, and emotional processing. Disruption to these circuits can lead to profound neuropsychiatric manifestations, including behavioural disinhibition. In this report, we describe the case of a 51-year-old male with a long-standing history of schizophrenia, who developed sudden onset hypersexuality and sexually inappropriate behaviour following a right basal ganglia infact. This behaviour change marked a dramatic deviation from his previous psychiatric baseline, which had remained stable for over a decade. To the best of our knowledge, this is the first documented case linking the right basal ganglia infarction with the emergence of hypersexuality in a patient with schizophrenia. We explore possible neurobiological mechanisms, particularly the alterations in inhibitory control and reward-processing pathways, and highlight the forensic and clinical implications of cerebrovascular lesions in modulating complex social behaviours.
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INTRODUCTION

Hypersexuality represents an under-recognised behavioural complication across a range of neurological disorders, with limited understanding regarding its phenotypic presentations, assessment strategies, and treatment approaches (1). Individuals with problematic hypersexual behaviour are unable to control their sexual cravings, regardless of other situational factors (2). Hypersexuality associated with neurological conditions remains poorly defined, despite its significant psychosocial consequences (1). Changes in sexual desire and behaviour are commonly observed following brain injury, primarily due to dysregulation of neural circuits involving the dopaminergic system, particularly the mesolimbic and mesocortical pathways (3). Organic brain disturbances, particularly related to subcortical areas, including the basal ganglia, may play a role in behavioural disinhibition and hypersexuality (4).

Clarifying the distinct features associated with particular neurological conditions could significantly advance pathogenesis models (1). Currently, no specific biomarkers exist that can reliably diagnose particular psychiatric or behavioural disorders, including those associated with sexual offending (5). This case illustrated a middle-aged man with the first case of sexual conduct towards children. He had been in the remission phase

of schizophrenia for many years with a 1-year history of increased sexual desire. This raised an alarm due to his newly found brain pathology, which was a basal ganglia infarct. He was sent to our institution for the assessment of criminal responsibility for the index sexual crime. We aimed to discuss possible neurobiological explanations of late-onset sexual behaviour, which resulted in sexual criminal behaviour following a cerebrovascular infarction.

CASE REPORT

A 51-year-old man with schizophrenia was admitted to a forensic ward for psychiatric evaluation following his first criminal offence, which was a sexual assault of two children. He admitted to kissing the girls and inserting a finger into one girl's private parts on the day of the incident, which occurred impulsively when he saw them playing outside his house. He was arrested on the same day. He expressed remorse and acknowledged the act was morally wrong but was unsure of the legal implications. He did not plan the offence and reported no auditory, visual, tactile, or gustatory hallucinations, delusions of persecution or control, or external influence. He recognized the act as his own.

He reported increased sexual desire over the past year, with him masturbating daily with a pillow, compared to his previous pattern of less sexual desire and infrequent masturbation. He did masturbation inside his house, usually inside his room. However, he never masturbated in public. He identified himself as heterosexual, single, with no prior love relationships. His sexual fantasies involved young females around 18 years old. He rarely viewed pornography due to limited access and had engaged in a single penetrative sexual encounter five years prior. He did not use sex workers or visit massage parlours due to financial constraints. The symptoms were not associated with headache, blurring of vision, double vision, or limb or facial weakness. He has no loss of appetite or weight loss.

He expressed a desire to marry, citing loneliness and the need for family. He believed marriage was difficult due to his lack of stable employment and social interest, which his sister confirmed. He had no history of interest in women his age or children. His self-esteem was reportedly high, though his sister denied this. He denied symptoms of mood disorder, anxiety, or obsessive-compulsive behaviours. His mental health had been stable for over 10 years, with no previous sexual misconduct or behavioural issues.

There were no symptoms suggestive of a mood disorder such as mania or hypomania, including elevated mood, decreased need for sleep, grandiosity, or increased goal-directed behaviour. The patient also did not exhibit cognitive deficits.

On examination, he appeared as a cooperative, well-groomed middle-aged male in hospital attire, with good rapport and eye contact. He described his mood as euthymic, with reactive affect, and showed no thought or perceptual disturbances. His judgment and insight were impaired, as he believed marrying an underage girl was neither legally nor morally wrong and showed a lack of empathy for the victims. A cognitive screening assessment using the Mini-Mental State Examination (MMSE) yielded a score within the normal range, supporting preserved global cognition.

Physical and laboratory assessments were unremarkable, including a normal neurological finding, except for a brain CT scan revealing an old infarct in the right basal ganglia. During a month of hospitalisation, he remained cooperative, independent in self-care, and engaged with staff and peers. His medication was continued with intramuscular fluphenazine decanoate 25 mg biweekly. The patient underwent regular psychiatric assessments to monitor behavioural control, risk of recidivism assessment, and insight evaluation regarding his sexual behaviours. Psychoeducation was provided to him, focusing on helping the patient to understand the legal boundaries of sexual conduct, develop victim empathy, and reinforce awareness of the consequences of sexual offences.

DISCUSSION

Individuals exhibiting problematic hypersexual behaviour are marked by a persistent inability to regulate sexual impulses, irrespective of contextual factors (2). Such behaviour can substantially complicate clinical management and negatively affect patient outcomes (3). Moreover, problematic hypersexuality may escalate into sexual offences, posing a major global concern due to the severe physical and psychological consequences suffered by victims (5). Although neurobiological mechanisms have been implicated in the pathogenesis of hypersexuality, definitive causal links have yet to be firmly established (1).

Hypersexuality has been identified across a range of neurological disorders, although its overall prevalence remains low, except in specific populations exposed to certain medications (1). Impaired control over sexual impulses is particularly evident in patients whose neurological conditions disrupt response inhibition mechanisms (2). A recent meta-analysis further highlighted that hypersexuality is a relatively common behavioural disturbance among individuals with neurological diseases, especially within neurodegenerative populations (3). Nevertheless, efforts to determine precise prevalence rates are limited by the absence of standardised, disorder-specific assessment instruments (1).

Clinically, manifestations of hypersexuality span a

spectrum from paraphilic behaviours to compulsive sexual activities, with notable qualitative differences across neurological conditions; for example, sexual compulsivity is more commonly observed in Parkinson's disease, whereas behavioural disinhibition is predominant in dementia syndromes (1). Individuals exhibiting hypersexuality consistently demonstrate poorer response inhibition compared to healthy controls, particularly when exposed to sexual stimuli (2). In cases where hypersexuality escalates to criminal behaviour, offences can range widely, including sexual assault, child sexual abuse, the production and distribution of child pornography, prostitution-related crimes, and female genital mutilation (5). While these behavioural patterns partially align with hypothesised underlying neuropathologies, robust comparative studies remain lacking (1). Although the body of research on hypersexuality continues to expand, much of the existing literature remains heavily focused on pedophilia, likely reflecting the intense societal concern surrounding sexual offences against children (5).

This case report describes a middle-aged man who, despite being in prolonged remission for schizophrenia, exhibited late-onset deviant sexual behaviour toward children following a newly identified basal ganglia infarct. His one-year history of escalating sexual desire raised clinical concern, prompting further investigation into potential organic causes. Previous case reports similarly underscore the role of the basal ganglia in behavioural disinhibition and deviant sexual conduct, particularly through its involvement in reward processing and inhibitory control (4). Importantly, the basal ganglia do not function in isolation; rather, they operate within broader neural networks, in coordination with neurotransmitter systems, to regulate behavioural inhibition and reward mechanisms. The basal ganglia interact with the inferior frontal gyrus (IFG), pre-supplementary motor area (preSMA), and limbic structures via fronto-striatal and mesocorticolimbic pathways. Disruption of these networks, particularly on the right side, can impair inhibitory control and amplify reward-driven impulses (2). Disruption of these circuits, as seen in this patient, can result in significant impairments in behavioural regulation, contributing to the emergence of disinhibited and socially inappropriate actions. Although the patient was in long-term remission from schizophrenia, the disorder may have contributed to baseline alterations in fronto-limbic and dopaminergic circuitry. Such pre-existing vulnerabilities could have lowered the threshold for behavioural disinhibition following the infarct. Moreover, chronic use of dopamine-blocking antipsychotics in this patient may induce receptor hypersensitivity or maladaptive plasticity, which may paradoxically fail to suppress, or even exacerbate, reward-driven behaviours in the presence of structural lesions.

Research into the neurobiological underpinnings of criminal behaviour represents a dynamic and expanding area within forensic neuroscience (4). Functional neuroimaging techniques, such as functional MRI (fMRI) and positron emission tomography (PET), have been utilised to explore patterns of brain activation during decision-making tasks and passive viewing of sexual stimuli, offering valuable insights into the neural mechanisms underlying sexual behaviours (5). Findings from these studies indicate that individuals with problematic hypersexual behaviour (PHB) exhibit reduced activation of the right inferior frontal gyrus (IFG) and weakened functional connectivity between the IFG and the pre-supplementary motor area (preSMA) during tasks requiring inhibitory control. Notably, these deficits in activation and connectivity are more pronounced when individuals are exposed to sexual distractors compared to neutral stimuli (2).

This case also raises important forensic considerations. The emergence of sexually inappropriate behaviour in a previously stable individual following a focal brain lesion highlights the need to consider neurobiological contributions in legal assessments. Forensic evaluations must account for the possibility that cerebrovascular damage may impair volition and moral reasoning, complicating determinations of criminal responsibility.

Lesion-induced criminal behaviour may result not only from focal damage to specific brain areas but also from disruption of broader neural networks involved in value-based and reward-driven decision-making (4). The mesolimbic pathway plays a central role in reward learning, while the mesocortical pathway is critical for executive decision-making processes. Dysregulation within these interconnected circuits is often clinically expressed as impulsive or compulsive behaviours, including manifestations of hypersexuality (3). Beyond structural and functional imaging findings, research into neurotransmitter systems has provided further insights into the biological mechanisms underpinning sexual offending, with particular attention to monoamine dysfunction and, more recently, the modulatory role of gamma-aminobutyric acid (GABA) in paraphilic disorders (5).

Beyond diagnosis, effective management of hypersexuality in patients with neurolo gical conditions remains a critical challenge (3). Treatment strategies should prioritise enhancing sexual self-regulation, mitigating problematic sexual behaviours and their adverse consequences, and addressing associated anxiety and functional impairments. To achieve these goals, patients require regular psychiatric follow-up, supplemented by vigilant monitoring from family members and relevant authorities (3, 4). Current evidence supports the use of combined pharmacological and non-pharmacological approaches as the most effective management strategy; however, specific treatment guidelines tailored to hypersexuality

secondary to neurological disorders are still lacking (3). Moving forward, the development of evidence-based protocols should be a research priority, necessitating multidisciplinary collaboration across the fields of neurology, neurosurgery, and psychiatry (1).

CONCLUSION

This case report illustrates how a right basal ganglia infarction can contribute to the late onset of deviant sexual behaviour through disruption of neural circuits governing inhibition and reward processing. The findings emphasise the need to consider organic brain pathology in forensic evaluations of sexual offences, particularly in individuals with previously stable psychiatric conditions. As forensic neuroscience advances, greater understanding of the neurobiological mechanisms underlying hypersexuality will be critical to improving diagnosis, management, and legal assessments. Future research should prioritise developing standardised diagnostic tools and evidence-based interventions to better address hypersexuality in neurological populations.

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REFERENCE

1. Tayim N, Barbosa P, Panicker J. Hypersexuality in neurological disorders: A systematic review. BMJ Ment Health. 2024;27(1). doi: 10.1136/bmjment-2024-300998
2. Seok J-w, Sohn J-H. Response inhibition during processing of sexual stimuli in males with problematic hypersexual behavior. Journal of Behavioral Addictions. 2020;9(1):71-82. doi: 10.1556/2006.2020.00003
3. Latella D, Maggio MG, Andaloro A, Marchese D, Manuli A, Calabrт RS. Hypersexuality in neurological diseases: do we see only the tip of the iceberg? Journal of Integrative Neuroscience. 2021;20(2):477-87. doi: 10.31083/j.jin2002051
4. Balcioglu YH, Dogan M, Incı I, Solmaz M. Sexual Behavioral Disinhibition Associated with Nucleus Lentiformis Lesion: A Forensic Neuroscience Perspective Through a Case. Journal of Forensic Sciences. 2020. doi: 10.1111/1556-4029.14477
5. Boschetti A, Ciani AC, Scarpazza C. Sexual offenses and the brain. Handbook of Clinical Neurology. 2023;197:161-79. doi: 10.1016/B978-0-12-821375-9.00017-7