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The Pharmacophilic Anchor: Compulsive Drug-Seeking Behavior as an Identity Tool in Post-Surgical Temporal Lobe Epilepsy

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Abstract

This case report explores the complex neuropsychiatric trajectory of "Noah," a 16-year-old adolescent with a history of drug-resistant lesional temporal lobe epilepsy (TLE) and severe childhood adversity. Despite achieving seizure freedom following a right anteromesial temporal lobectomy, the patient experienced progressive cognitive decline and the development of an organic-based affective disorder with borderline traits and gender dysphoria. A defining feature of the clinical presentation is a "pharmacophilic" pattern: a compulsive search for a transformative pharmacological solution, characterized by repeated therapy escalations and paradoxical behaviors, such as self-induced vomiting following drug ingestion. The discussion integrates neurobiological dysfunction of mesial temporal structures, critical nodes for reward processing and emotional regulation, with the "burden of normality" framework and environmental trauma. Noah's clinical course illustrates a significant dissociation between neurological and psychiatric outcomes, where pharmacological intervention assumed a symbolic role for relational manipulation and identity management. This case underscores the limitations of purely neurological models and advocates for a multidimensional approach in TLE management. Addressing the interaction between damaged neural substrates, developmental vulnerabilities, and iatrogenic reinforcement is essential to manage enduring emotional dysregulation and maladaptive chemical expectancies in pediatric populations.

Keywords: Temporal Lobe Epilepsy (TLE); Pharmacophilia; Post-surgical outcomes; Gender dysphoria; Burden of normality; Identity dysregulation; Drug-seeking behavior; Cognitive erosion

Introduction

Resective surgery represents a cornerstone in the management of drug-resistant focal epilepsy, particularly in pediatric populations, where it may offer the only chance for long-term seizure freedom [1]. Beyond seizure control, however, increasing attention has been directed toward the broader neuropsychiatric and developmental consequences associated with temporal lobe epilepsy (TLE) and its surgical treatment. The mesial temporal region, including the amygdala and hippocampus, plays a crucial role not only in memory but also in reward processing, emotional regulation, and decision-making [2]. These functions rely on a distributed neural network involving the orbitofrontal cortex, ventromedial prefrontal cortex, striatum, anterior cingulate cortex, and limbic structures [3]. Disruption of this circuitry, as observed in TLE, may alter feedback processing, reward sensitivity, and motivational behaviors, ultimately impairing adaptive decision-making. In parallel, emotional processing depends on large-scale brain networks such as the salience network, default mode network, and central executive network, which support the detection of relevant stimuli, self-referential processing, and cognitive control [4]. Neurodevelopmental evidence suggests that these systems continue to mature throughout adolescence, making this period particularly vulnerable to the effects of neurological insults [5]. Importantly, TLE is associated with significant impairments in social cognition, including theory of mind and emotion recognition, with large effect sizes reported across studies [6]. These deficits, particularly pronounced in right

temporal involvement, may contribute to difficulties in interpersonal functioning and emotional understanding. Furthermore, epilepsy is strongly associated with psychiatric comorbidities, including depression, anxiety, irritability, and atypical affective presentations, often resistant to standard diagnostic categorization [7]. While surgery may improve some emotional domains, psychiatric outcomes remain heterogeneous and not necessarily aligned with seizure control [8]. Taken together, these findings suggest that alterations in mesial temporal structures may have profound and multidimensional consequences, particularly during development. The present case illustrates how neurological vulnerability, emotional dysregulation, and maladaptive behavioral patterns may converge, giving rise to complex clinical presentations that extend beyond traditional neurological or psychiatric frameworks.

Case Report

The clinical case involves Noah, a 16-year-old adolescent assigned female at birth, who has been under the care of the Child and Adolescent Neuropsychiatry service (ASL CN2). Noah's history is rooted in drug-resistant focal lesional epilepsy secondary to a low-grade glioneuronal lesion in the right uncus-amygdala region, for which he underwent a right anteromesial temporal lobectomy in September 2012. Although the neurological framework eventually led to a slow, progressive remission and seizure freedom, this "success" was paradoxically paralleled by a troubling cognitive erosion, with his total IQ dropping from 77 in 2017 to 62 in 2023. This decline in cognitive resources significantly impaired Noah's ability to process a deeply traumatic family environment, marked by maternal anxious-depressive symptoms, paternal alcoholism, physical violence, and a history of sexual abuse. Within this context of structural and cognitive fragility, Noah developed intense gender dysphoria; identifying as gender-fluid, he expressed a persistent aversion to his body and consistently requested a mastectomy. These factors, the compromised neural substrate, the intellectual decline, the unresolved identity conflict and the traumatic experiences, converged to create a profound "identity-related void". In the absence of internal psychological tools to manage this distress, Noah developed a marked pharmacophilic attitude. His organic instability fostered a chronic, misplaced expectancy that a definitive chemical solution could compensate for both his neurological "damage" and his bodily estrangement. Consequently, the pharmacological act evolved into his primary tool for relational manipulation and self-regulation. This pattern became evident in his treatment evolution: by 2013, the initial regimen of topiramate and carbamazepine transitioned to levetiracetam and oxcarbazepine. By 2019, following the onset of panic attacks, the therapy integrated psychiatric agents such as pregabalin and quetiapine. As behavioral worsening and dietary restrictions emerged in 2021, Noah began relying on diazepam as needed. By January 2022, reported "voices in the head" led to further escalations of oxcarbazepine and levetiracetam. Following a pediatric hospitalization in April 2022, aripiprazole and lorazepam were introduced. However, the baseline neurological distress remained a constant catalyst for further requests. By December 2022, Noah's pharmacophilia culminated in a high-risk overdose of lorazepam, an event that signaled the failure of his previous multi-drug regimens. This crisis was immediately followed by the onset of daily induced vomiting post-ingestion. This behavior established a paradoxical clinical framework: a compulsive, drug-seeking drive for a biological anchor to ground his insatiable identity, coexisting with the active sabotage of the very molecules he demanded. The therapeutic framework was radically overhauled in March 2023, transitioning to a combination of lamotrigine, lithium sulfate, aripiprazole, and pregabalin. Because the new therapeutic shift proved insufficient against his recurring behavioral crises, quetiapine was repeatedly titrated in an attempt to achieve sedation. By late 2023, the pharmacological landscape had expanded into a complex combination of high-dose lamotrigine and quetiapine, layered with a rotating sequence of benzodiazepines (delorazepam, lorazepam, alprazolam, bromazepam, diazepam) and chlorpromazine. Given the failure to achieve stabilization at home, Noah was admitted to a specialized rehabilitation facility. Even within this structured environment, his course remained punctuated by ER admissions triggered by acute behavioral dyscontrol, fostering a self-perpetuating cycle of therapy augmentation. In early 2024, the onset of hyperprolactinemia necessitated a re-evaluation of the pharmacological burden. This side effect carried significant psychological weight, as the potential progression toward galactorrhea threatened to physically exacerbate his gender dysphoria. Leveraging a trusting therapeutic relationship with his clinical team, Noah agreed to a gradual reduction of chlorpromazine in favor of a nutraceutical component containing Griffonia, Magnesium, Zinc, and L-theanine. This strategic shift represented a deliberate attempt to alleviate the cumulative antipsychotic and benzodiazepine loading while maintaining a stable biological framework. Nevertheless, the underlying quest for a "chemical savior" persisted. In May 2025, after voluntarily ingesting degreaser, he requested a new hospitalization specifically to alter his therapy once more. A similar event occurred again in February 2026 with the incongruous ingestion of moisturizing cream, followed by pressing demands for medication increases. As of March 2026, the clinical landscape remains a deadlock of chemical expectancies: the interaction between a compromised neurological substrate and a fragile psychological identity continues to drive a relentless search for a transformative pharmacological savior, currently reflected in a complex multi-

drug regimen consisting of: Lamotrigine (100 mg/die), Lithium Sulfate (83 mg/die), Chlorpromazine (100 mg/die), Zolpidem Tartrate (10 mg/die), Alprazolam (2.5 mg total/die), Quetiapine (100 mg/die), Sertraline (100 mg total/die), Clonazepam (2 mg/die), and Bromazepam (3 mg/die).

Discussion

Noah's clinical presentation reflects a complex intersection of structural brain damage, altered neurocognitive processing, and severe environmental adversity, which resulted in a highly atypical and multifaceted phenotype. In this case, the right uncus-amygdala lesion and subsequent temporal lobectomy fundamentally compromised a key node in the brain's decision-making and reward network [9,10]. The disruption of these systems likely impaired the ability to appropriately evaluate costs and benefits, leading to a persistent overvaluation of anticipated pharmacological effects despite a history of therapeutic failures. Notably, this pattern persisted even after achieving seizure remission, suggesting it was not merely a reactive response to active epilepsy but rather reflected an enduring alteration in reward expectancy and decision-making processes. This mechanism helped explain the emergence of a pharmacophilic pattern characterized by compulsive drug-seeking behavior. Interestingly, this drive was paradoxical, as it coexisted with behaviors that actively interfered with drug efficacy (e.g. purging) suggesting a deep dysregulation of the motivational system rather than a standard dependency model [11,12]. Beyond reward processing, deficits in social cognition further complicated the clinical picture. TLE had been consistently associated with impairments in Theory of Mind and emotion recognition, with large effect sizes reported across multiple paradigms [6]. Such deficits may have limited the patient's capacity to interpret social cues, understand the intentions of others, and regulate interpersonal interactions, thereby contributing to relational instability and the adoption of maladaptive behavioral strategies.

Consistent with this framework, TLE was associated with a high prevalence of mood and anxiety disorders that often manifested in atypical forms, including irritability, dysphoria, and fluctuating affective states [7]. The dissociation between neurological and psychiatric outcomes was particularly evident here: while seizure freedom had been achieved, severe emotional and behavioral dysregulation persisted. Surgical intervention does not guarantee psychiatric improvement; while anxiety may decrease post-operatively, depressive symptoms often endure or, in some instances, worsen [13]. Developmental factors further add to this complexity, as it is with Noah's case, pediatric epilepsy populations exhibit high rates of neurodevelopmental and psychiatric comorbidities, including ADHD, autism spectrum traits, and intellectual impairment, which lead to significant heterogeneity in long-term outcomes [14]. From a psychological perspective, the concept of the "burden of normality" is central to understanding Noah's trajectory. Following surgery, patients often struggle to adapt to new roles and expectations, particularly when the anticipated life improvements fail to materialize [7]. Noah's difficulty in transitioning from a chronic illness identity to a new, less defined condition fitted this framework and likely fueled his persistent search for a "transformative" pharmacological solution. Crucially, environmental factors played a significant amplifying role; severe trauma, neglect, and family instability had likely interacted with his neurobiological vulnerabilities to foster borderline traits, identity disturbances, and maladaptive coping mechanisms. Within this volatile context, pharmacological treatment assumed a symbolic and relational function, serving as a tool for communication, control, and the management of internal distress. Finally, Noah's clinical trajectory raises critical considerations regarding iatrogenic processes. Repeated emergency interventions and frequent pharmacological adjustments may have reinforced the maladaptive belief that stability could only be achieved through medication changes or dosage escalation. This was particularly concerning given that his seizures were no longer present; continued pharmacological escalation may have reinforced the conviction that his internal instability was exclusively biologically driven, requiring an endless cycle of new chemical interventions.

Conclusions

This case underscores the importance of a multidimensional approach to patients with TLE, integrating neurological, psychiatric, developmental, and environmental factors. Pharmacophilic behaviors, as observed in this patient, may represent the behavioral expression of deeper disruptions in reward processing, emotional regulation, and identity formation. The persistence of pharmacophilic behavior in the absence of active seizures highlights the need to move beyond a purely neurological model and adopt an integrated framework that addresses enduring alterations in neurocognitive, emotional, and relational domains. Future management should aim to reduce iatrogenic reinforcement, enhance psychological containment, and promote more integrated and sustainable forms of regulation.

Ethics approval and consent to participate

Ethical committee approval was not required for this case report, as it involved a retrospective review of a single patient with all identifying information removed to ensure confidentiality. Informed consent was obtained for the publication of this case report and the clinical data involved.

Data Availability Statement

The data that support the findings of this case report are available from the corresponding author, upon reasonable request. The data are not publicly available due to privacy or ethical restrictions involving patient clinical information.

Conflict of interest

The authors declare no conflict of interest.

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