

Case Report

The paradox of care: dopamine super sensitivity psychosis and catatonia following excessive antipsychotic exposure in an adolescent with first-break schizophrenia

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ABSTRACT

Dopamine super sensitivity psychosis (DSP) is marked by rebound or treatment-resistant psychosis, typically linked to prolonged or poorly managed antipsychotic use. While increasingly recognized in adults, DSP in adolescents, especially with catatonia, remains underreported. The impact of fragmented psychiatric care and caregiver-driven treatment changes is rarely addressed. We describe a 13-year-old female with schizophrenia who developed severe functional decline and catatonia after years of inconsistent psychiatric management. Extensive workup, including EEG, brain MRI, and broad metabolic, infectious, autoimmune, and endocrine testing, was negative. Her history revealed frequent physician changes and abrupt medication switches without tapering, often initiated by parental concern over persistent symptoms. At presentation, she was wheelchair-bound, minimally responsive, and showed psychomotor retardation, tremors, and auditory hallucinations. Her course aligned with DSP complicated by catatonia, after incomplete or subtherapeutic trials of risperidone, olanzapine, quetiapine, aripiprazole, and cariprazine. Treatment with clozapine and high-dose lorazepam was initiated, alongside careful monitoring and structured support for the family. The patient gradually regained independence, resumed psychotherapy, and re-engaged socially, with significant improvement in psychosis. This case illustrates the rare intersection of adolescent DSP and catatonia, highlighting risks of inconsistent treatment and caregiver-driven decisions. It underscores the importance of coordinated psychiatric care, cautious medication adjustments, and comprehensive family education. Supporting caregivers emotionally and educationally is vital to improving adherence and long-term outcomes for youth with severe mental illness.

Keywords: Dopamine super sensitivity psychosis, Catatonia, Antipsychotic, Schizophrenia, Clozapine

INTRODUCTION

Schizophrenia is a chronic and often disabling mental illness that globally remains one of the top 10 leading causes of disability, emphasizing the need for empathetic and effective long-term care.¹ Although antipsychotics are effective in managing the acute symptoms of schizophrenia, it is important to recognize that improper switching or abrupt discontinuation of medication without proper tapering can result in serious consequences.²⁻⁴ One significant risk is the development of dopamine super sensitivity psychosis, which is linked to neuroadaptations, such as dopaminergic hypersensitivity that persists after stopping antipsychotic treatment, underscoring the need for gradual tapering to minimize relapse and withdrawal effects.²⁻⁵

Dopamine super sensitivity psychosis (DSP) is a state in which patients, typically with schizophrenia, develop unstable or rebound psychosis after long-term or high-dose antipsychotic treatment.^{6,7} The main manifestations include new or worsening psychosis episodes despite ongoing or increased antipsychotic dosing, tolerance to antipsychotic effects, and tardive dyskinesia, often triggered by dose reduction, withdrawal, or switching of antipsychotics.^{2,3} In recent years, researchers have begun to take a closer look at the idea of iatrogenic dopamine super sensitivity psychosis (DS), with growing recognition of it as a real and pressing clinical concern.⁸

Catatonia is a neuropsychiatric syndrome and its diagnosis is typically based on the presence of at least three of twelve key symptoms, including stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation, grimacing, echolalia, and echopraxia, as outlined in the DSM-5.^{9,10} Abrupt discontinuation or long-term use of antipsychotics, especially those affecting dopamine pathways, can precipitate catatonia, suggesting it may be a manifestation of dopamine super sensitivity psychosis that is often overlooked in clinical practice.^{11,12} Additionally, studies have supported that catatonia is an underreported presentation of dopamine super sensitivity psychosis, highlighting the complex interplay between dopaminergic dysfunction and catatonic symptoms.^{10,11,13}

Schizophrenia that begins during adolescence often follows a more severe clinical course, with significant functional decline and a higher risk of poor long-term outcomes.¹⁴ The management of adolescent-onset schizophrenia presents profound clinical and emotional challenges, both for the patient and their caregivers. When psychosis persists or recurs despite treatment, families, often overwhelmed by fear and the desperation for the cure may pursue multiple psychiatric providers and treatment options resulting in frequent medication changes. In such cases, well-intentioned but urgent pharmacological interventions, often involving repeated antipsychotic switches or dose adjustments without proper tapering and monitoring, can inadvertently lead to

treatment resistance and dopamine super sensitivity psychosis (DSP). Although DSP is increasingly recognized in adults with treatment-resistant schizophrenia, its manifestation in adolescents remains sparsely documented and its roots are rarely examined in the context of caregivers. Here, we bring to attention an infrequently documented aspect of adolescent schizophrenia, while contributing to the rare presentation of DSP with catatonia, and highlight how parental desperation may influence psychiatric care and contribute to the development of dopamine super sensitivity psychosis.

CASE REPORT

A 13-year-old girl with treatment-resistant schizophrenia and rebound psychosis was brought to the clinic. On initial presentation, the patient was seated immobile in a wheelchair, showing severe psychomotor slowing and stupor, characterized by marked unresponsiveness, immobility, and absence of spontaneous activity. She gave little to no verbal response, consistent with mutism, and her facial expression remained fixed and unchanging throughout the encounter. Despite multiple attempts to engage her, she did not initiate movement, conversation, or eye contact, and appeared largely disconnected from the surroundings. She also demonstrated passive negativism, as she consistently failed to respond or move despite verbal prompts. There was an absence of purposeful activity, and she showed no spontaneous interaction with the environment or with us.

She exhibited drooling and intermittent tremors. According to her parents, she had become nearly entirely dependent on activities of daily living, including feeding and dressing. They also reported that she had grown increasingly emotionally withdrawn, disorganized, and distractible in recent months, with a marked deterioration in the weeks preceding admission. The patient endorsed auditory hallucinations, describing a persistent and distressing male voice she identified as a “bad devil,” which accused her of “sinning” and commanded her to pray to an alternative deity. These experiences were accompanied by progressive fearfulness, social withdrawal, and cognitive disorganization, raising concern for an evolving psychotic process with catatonic features.

Her psychiatric history was notable for early-onset schizophrenia and fragmented care. She was initially evaluated at age 10 for persistent low mood, frequent crying spells, anxiety, and auditory hallucinations. However, her treatment was inconsistent, frequently interrupted by changes in providers or family-initiated discontinuation out of concern for limited or slow improvement. Over the subsequent three years, various psychotropic medications were trialled, often without gradual tapering or adequate monitoring, contributing to a pattern of episodic partial remissions followed by relapse

psychosis. Medication records were limited, but the family narrative highlighted a lack of continuity in care.

Given her poor response to prior treatments, the severity of her symptoms, and parental reluctance regarding electroconvulsive therapy (ECT), she was started on clozapine 25 mg twice daily and lorazepam 6 mg per day. At the time of admission, the family was visibly distressed, overwhelmed by the chronicity and unpredictability of her illness course. Recognizing the emotional burden, the clinical team prioritized transparent communication and emotional support, educating the family about dopamine super sensitivity psychosis (DSP), potential side effects, and long-term management strategies. Space was created for the family to voice their uncertainty, guilt, and frustration, emotions accumulated over years of fragmented psychiatric care.

As rapport improved, the family became actively engaged in her treatment. Follow-up visits became consistent, and coordinated care was established. Over the following weeks, the patient demonstrated steady improvement. By her most recent evaluation, she was walking independently, no longer reliant on a wheelchair, and actively participating in psychotherapy. She also began engaging in volunteer work and reported reduced auditory hallucinations.

DISCUSSION

Dopamine super sensitivity psychosis (DSP) is a condition in which symptoms such as hallucinations and delusions reemerge or worsen despite ongoing or escalating doses of antipsychotic medication. This is thought to result from upregulation and increased sensitivity of dopamine D2 receptors following prolonged antipsychotic use.^{15,16} Several possible reasons can lead to dopamine super sensitivity psychosis (DSP), including long-term antipsychotic treatment, abrupt discontinuation, dose changes, and the development of tolerance to antipsychotics.^{2,17,18} Clinical manifestations such as tardive dyskinesia, rebound psychosis, and tolerance are recognized as manifestations of DSP.¹⁷ Catatonia has also been reported as a possible presentation of DSP describing the emergence of catatonic symptoms in patients with a history of long-term antipsychotic treatment and dopamine super sensitivity.^{11,19}

Child or adolescent-onset schizophrenia often presents with severe negative symptoms, bizarre and impulsive behaviours, and is associated with a higher risk of poor long-term outcomes, including persistent functional impairments and social deficits.^{20,21} These patients may be more likely to require long-term, excessive polypharmacy, which increases the risk of developing dopamine super sensitivity psychosis (DSP) due to prolonged and complex antipsychotic exposure.

Aside from the extensive use of the antipsychotics, the utilization of multiple different antipsychotics brings up

complications and issues. As previously mentioned, DRD2 receptors can be upregulated as a result of multiple antipsychotic usage, but dopamine receptors are not the only receptors that can be upregulated due to psychiatric medication usage. Multiple receptor subtypes such as 5-HT_{2A}, M₁, α_1 , and other receptors can become upregulated as a result of switching antipsychotics. If a patient switches antipsychotics too abruptly, there is a significant possibility that the antipsychotics will have varying affinities for neuroreceptors, which can cause the patient to experience withdrawal symptoms or a resurgence of psychosis.²² Once DSP occurs, these symptoms become easily exacerbated by a small reduction in antipsychotics. Recovering from excessive polypharmacy is extremely difficult for patients in a state of DSP.²³

Studies have shown that Clozapine, a second-generation (atypical) antipsychotic, can be considered a good choice for treating DSP as it can effectively alleviate the symptoms and reduce the recurrence of episodes of psychosis in patients who have not responded to other antipsychotics.²⁴⁻²⁶ Its unique pharmacological profile, including low affinity for dopamine D2 receptors and high affinity for serotonin 5-HT_{2A} receptors, is thought to help reverse dopamine super sensitivity and provide symptom relief in DSP cases.^{25,26}

Our case describes a rare presentation of dopamine super sensitivity psychosis (DSP) with catatonia in an adolescent, a combination that remains poorly documented in the current literature. The patient was referred to our care after experiencing repeated medication failures and escalating psychotic symptoms. Prior to presentation, her treatment involved frequent changes in psychiatrists, multiple antipsychotic trials without proper tapering, and inconsistent follow-up, all of which likely contributed to the emergence of DSP. At the time of referral, electroconvulsive therapy (ECT) had been recommended by a previous provider to whom the family expressed significant reluctance. Understandably overwhelmed and frustrated, they sought an alternative treatment plan. With this in mind, we initiated clozapine with lorazepam, closely monitored the patient's clinical response, and made a parallel effort to build trust with the family. We provided psychoeducation on DSP, addressed their concerns about treatment, and offered space to process the emotional struggle of their journey. Over time, as the family engaged more consistently with follow-up and became active participants in care, the patient began to show steady and significant improvement. Her catatonic symptoms resolved, her effect and speech gradually returned, and she started volunteering, a significant recovery that felt almost unimaginable at the outset.

CONCLUSION

While contributing to the limited literature on dopamine super sensitivity psychosis (DSP) presenting with catatonia in adolescents, we also aim to highlight a

critical, yet often overlooked, factor: the role of caregiver behavior in the treatment outcomes. In this case, the patient's clinical course was likely exacerbated by well-intentioned but discontinuous care, marked by frequent psychiatrist changes, abrupt medication switches without proper tapering, and inconsistent follow-up. These actions, often driven by desperation and fear, can unintentionally worsen the trajectory of illness, especially in vulnerable youth patients.

We hope to highlight the need for early, clear communication with families about the potential risks associated with rapid or uncoordinated treatment changes, including the development of DSP. Beyond clinical management, supporting caregivers emotionally and helping them understand the effects of their behaviour on a child's condition are essential. With emotional support, clear guidance and continuity, families can become our collaborative allies in navigating the complexities and challenges of adolescent schizophrenia, ultimately improving both treatment compliance and outcomes.

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