

# From Psychedelic Exploration to Psychosis: A Unique Case Study Demonstrating the Susceptibility to Schizophrenia Unveiled by a Single LSD Experience

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## Abstract

Lysergic acid diethylamide (LSD) is part of the pharmacological group known as “classical hallucinogens” or “psychedelics” (a term coined by Humphrey Osmond in 1957) while sharing its chemical structure with psilocybin and dimethyltryptamine <sup>[1]</sup>. Though the exact mechanism is unclear, classic hallucinogens are believed to work mainly as agonists at the serotonin 2A (5-HT<sub>2A</sub>), 2C (5-HT<sub>2C</sub>), and 1A (5-HT<sub>1A</sub>) receptors, as well as producing effects in the dopaminergic and noradrenergic systems <sup>[2]</sup>. These hallucinogens carry some risk. One of them includes a dangerous state of anxiety and psychosis, causing unpredictable behaviors in an uncontrolled environment. Another possible risk is the exacerbation of psychotic disorders <sup>[3]</sup>. This case depicts a previously healthy young female who presented with a severe case of LSD-induced psychosis with a cataonic-like state. We summarized her hospital course, provided extensive medical workup, and underwent several psychopharmacology trials with the initiation of electro-convulsive therapy that eventually led to her recovery. Our case illustrated the importance of identifying the adverse side effects of LSD and its risk of lowering one’s threshold for developing schizophrenia. “AD” is a pseudonym and identifying information was removed to protect the patient’s confidentiality.

## Keywords

LSD, LSD-Induced Psychosis, Acute Psychosis, Catatonia, Electro-Convulsive Therapy, Case Report

## CASE

Patient “AD” was a previously healthy 29-year-old African-American female who presented to our inpatient psychiatric facility for psychosis and catatonic symptoms. AD was originally involuntarily admitted to another hospital three weeks prior for suicidal ideation. However, psychotic symptoms failed to improve, so she was transferred to our facility for consideration of electroconvulsive therapy (ECT).

On presentation to our facility, AD was a poor historian. She was not orientated to person, time, or place and appeared paranoid and agitated about her surroundings. She was floridly psychotic and was responding to internal stimuli while mumbling to herself. She made repetitive phrases such as “imposter syndrome” and “Whiskey Mike” without any

elaboration. She was religiously preoccupied, making statements of “King Jesus” and writing religious phrases in her notebook. Her speech was disorganized, tangential, and incoherent with periods of thought blocking. Additionally, she seemed sexually preoccupied and was seen following male individuals around the inpatient unit while trying to take her clothes off. On exam, she exhibited catatonic symptoms such as rigid posture, waxy flexibility, posturing, agitation, and stereotypy.

Her family reported that prior to this incident, AD was a well-mannered, soft-spoken individual with no history of any inappropriate behaviors, aggression, or psychotic episodes. Her family history was negative for any psychotic conditions. According to AD’s mom, AD had smoked cannabis laced

with Lysergic acid diethylamide (LSD) provided by her boyfriend days prior to admission. Details of this incident were unknown. As days progressed, her symptoms started to manifest and worsen exponentially, so much so that her mother stated that AD became “unrecognizable.”

Due to the abrupt onset of symptomatology, our initial goal was to rule out any organic causes or conditions of autoimmune origins. Her urine drug screen was positive for marijuana, and her blood alcohol level was unremarkable. Her urinalysis showed acute cystitis, which was treated with ciprofloxacin. Her thyroid stimulating hormone and complete metabolic panel were within normal limits.

AD was evaluated with a head CT without contrast and an MRI brain with contrast, both of which showed benign findings. In the following weeks of her admission, she failed multiple trials of antipsychotics such as Risperdal and Zyprexa. Risperdal caused worsening of her catatonia, and Zyprexa was completely ineffective even at the maximum recommended dose. Geodon was initiated, which showed a slight improvement in symptoms. Neurology was consulted to rule out any possible neurological deficits and autoimmune conditions. Subsequently, AD underwent an EEG that showed no signs of stroke, seizure activity, or any abnormalities in brain function. Her lumbar puncture showed no evidence of CNS infections and autoimmune processes. Additionally, due to AD’s rapid manifestation of neuropsychiatric changes, there was concern for anti-NMDA receptor encephalitis [4]. A full autoimmune encephalitis panel including ANCA, Anti-cardiolipin Abs, NMDA, Proteinase-3 Ab, Sjogren’s Abs, Smith Abs, and SclAb was conducted, but the results were negative.

Throughout her hospitalization, it became clearer that AD’s psychosis was worsening. She demonstrated increasingly self-destructive behaviors such as banging her head on

furniture and scratching herself. Additionally, she was physically aggressive to her peers and became sexually aggressive towards them. In one instance, she believed one of her peers was her husband. With her increasing psychosis and catatonic-like symptoms, we ultimately decided to pursue electroconvulsive therapy (ECT). After her first two treatments, AD’s clinical signs started improving drastically. She became more aware of her surroundings, started to remember the names of staff and peers, and regained self-awareness of who she previously was. Her judgment and insight improved and she started apologizing for weeks of inappropriate behaviors. Her symptoms of catatonia slowly resolved throughout the course of ECT. After five treatments of ECT, her symptoms had almost completely resolved, and she was discharged to follow up with outpatient ECT treatments.

## **DISCUSSION**

With the increasing research on psychedelics for the treatment of psychiatric conditions, it is also important to note the potential side effects of these substances. LSD, taken at moderate doses, produces changes in body perception, time distortion, and psychotropic effects, often described as “mystical experiences.” These effects can mimic a psychotic-like state for a few hours. However very rarely, this may induce a much longer-lasting psychosis, like AD [5]. While LSD is currently a Schedule I drug and not FDA-approved, research has shown that LSD-assisted psychotherapy may positively impact patients suffering from illness-related anxiety, depression, psychosomatic conditions, substance abuse, and cancer-induced pain disorders [6]. Oral doses of LSD have provided long-term improvements in patients’ attitudes about life or self and positive mood changes [7]. In studying the effects of oral LSD and changes in brain structure, neuroimaging researchers found a significant decrease in left

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amygdalar reactivity to fearful stimuli as compared to controls, implying that LSD may be useful in reducing perceptions of negative emotions in depressive disorders [8].

This case report adds to the growing literature related to LSD-induced psychosis and catatonia with a previously healthy patient without a known family history of psychotic disorders. Breakey et al. conducted a comprehensive history of 46 schizophrenic patients with 46 matched controls. They discovered that schizophrenics who had used hallucinogens experienced the onset of psychosis on average four years earlier than non-users [9]. Another survey conducted in the 1950s found that one individual out of 1200 participants experienced prolonged psychosis after using hallucinogenic drugs. Notably, that individual has a family history of a twin brother with schizophrenia [10]. These data demonstrated that there is evidence of hallucinogens such as LSD that may play a role in lowering the threshold for the development of schizophrenia. Though rare, these adverse effects exist. This case demonstrates the importance of education and early treatments to improve patients' prognosis and quality of life.

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