LSD-Induced Catatonia in an Adolescent:

A Rare Presentation With Clinical Implications for Primary Care

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Catatonia is a complex neuropsychiatric syndrome marked by a range of abnormal motor behaviors, behavioral disturbances, and withdrawal. It poses a diagnostic challenge in clinical practice because it can occur across a wide spectrum of psychiatric, medical, and neurological conditions.[1]

As defined in the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition, a diagnosis requires at least 3 characteristic features, which may include stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation unrelated to external stimuli, grimacing, echolalia, and echopraxia.[2] Reported prevalence in acute psychiatric settings ranges from 9% to 18%,[3,4] and approximately 16% of psychiatric inpatients meet diagnostic criteria.[5] Incidence estimates from liaison psychiatry services vary by age group and clinical setting, ranging from 1.6% to 8.9%.[6,7]

While historically catatonia was ascribed primarily to psychiatric origins, contemporary research highlights a more expansive etiological landscape encompassing medical conditions such as neoplasms, paraneoplastic syndromes, metabolic disorders, infections, drug exposures, and poisoning incidents.[8,9] Recognition of substance-related presentations is especially important in primary care and emergency settings, where timely diagnosis can facilitate effective intervention. This report describes a case of catatonia associated with lysergic acid diethylamide (LSD) use. LSD is available on the streets with different names including "acid," "microdots," mellow yellow," "sunshine," "zen," and "windowpanes."[10] LSD was first synthesized in 1938 and gained prominence in the mid-1960s for its potent hallucinogenic properties, evident at doses as low as 25 µg.[11,12] Epidemiological data from 2002 to 2019 indicate a consistent rise in LSD use across all age groups, including individuals aged 12–17, 18–25, and ≥26 years.[13] In 2023, roughly 8 million individuals aged 12 years or older reported recent use of hallucinogens, including LSD, underscoring its continued relevance in contemporary substance use and the potential for severe psychiatric sequelae.[14]

Case Report

A 16-year-old boy presented to the emergency department (ED) with a 1-day history of markedly reduced oral intake, social withdrawal, and mutism. His past medical history was notable for marijuana use disorder. On examination, he demonstrated rigidity in multiple extremities and was unable to follow simple commands. Mutism was also observed, consistent with a catatonic presentation. Collateral information from family members confirmed the history of reduced oral intake and social withdrawal. At admission, his vital signs were within normal limits, with a blood pressure of 120/80 mm Hg, heart rate of 80 beats/minute, respiratory rate of 16 breaths/minute, and an afebrile temperature of 98.6°F (37°C).

Given the presentation suggestive of catatonia, the pediatric ED physician, in collaboration with the psychiatry team, administered a diagnostic and therapeutic trial of intravenous lorazepam 2 mg. The patient demonstrated a marked improvement in symptoms within 45 minutes of administration, supporting the diagnosis of catatonia. He was subsequently admitted to the inpatient pediatric unit for further evaluation and management. Initial investigations included a noncontrast computed tomography scan of the head and lumbar puncture, both of which were unremarkable. Urine toxicology screening was positive for cannabinoids, confirming the presence of urinary tetrahydrocannabinol. Additional laboratory tests showed normal electrolyte values (phosphorus: 2.8 mg/dL; magnesium: 2.3 mg/dL), liver function tests within normal limits (aspartate aminotransferase: 12 U/L; alanine aminotransferase: 12 U/L; alkaline phosphatase: 133 U/L), and a complete blood count within reference ranges.

The patient was maintained on oral lorazepam 2 mg 3 times/d for catatonia management. His care was coordinated through a multidisciplinary team involving neurology, psychiatry, and pediatrics. During hospitalization, he developed new-onset auditory hallucinations and paranoid ideation, indicating progression to psychosis. Olanzapine orally disintegrating tablets were initiated at 5 mg/d and later titrated to 10 mg/d, with gradual improvement in psychotic symptoms.

Further diagnostic evaluation was undertaken to exclude organic causes. Magnetic resonance imaging of the brain revealed an incidental Rathke cleft cyst, and anti-*N*-methyl-D-aspartate receptor antibody testing was negative, making autoimmune encephalitis unlikely. With organic causes reasonably excluded, alternative etiologies were considered. Upon obtaining a more detailed history, the patient disclosed recent

ingestion of LSD in the form of a blue gel shortly before symptom onset. Family confirmation, together with a review of the patient's cellphone revealing text exchanges explicitly arranging LSD purchase and use, provided strong corroboration of recent LSD ingestion. The absence of any preceding primary psychiatric episode, such as depression, mania, psychosis, or prodromal symptoms, further reinforced the likelihood of LSD-induced catatonia.

Catatonia and psychosis persisted despite pharmacologic treatment. As electroconvulsive therapy (ECT) was unavailable at the current facility, the team arranged transfer to an inpatient pediatric psychiatric unit with ECT capability. After 18 days of hospitalization and multidisciplinary management, the patient was transferred to the designated psychiatric facility for continued treatment, including the potential initiation of ECT for refractory catatonia and psychosis.

Discussion

The present case highlights a potential association between LSD use and catatonia, emphasizing the diagnostic complexity of substance-induced catatonia in the context of limited research and clinical awareness. Catatonia itself carries significant morbidity and often requires inpatient medical or psychiatric care to prevent complications such as deep vein thrombosis, pulmonary embolism, decubitus ulcers, muscle contractures, and rhabdomyolysis leading to renal failure.[15] Previous studies, including case series, systematic reviews, and meta-analyses, have documented an association between catatonia and substance intoxication or withdrawal.[16,17] Several reports describe catatonia linked to cannabis or synthetic cannabinoid use.[18] However, there is a notable paucity of literature specifically addressing LSD-associated catatonia. Given that LSD primarily acts as a potent agonist at central serotonin 5-HT_2 receptors, which are implicated in various psychiatric syndromes, it is biologically plausible that LSD could serve as a trigger, if not a direct cause, of catatonic states.[19] In this case, the patient ingested LSD in the form of a blue gel, which has been reported to be a more concentrated formulation than certain other preparations and may have contributed to the severity of his neuropsychiatric presentation. At higher doses, LSD's dopaminergic and adrenergic activity may also contribute to adverse neurological and psychiatric effects.[20] Despite ongoing debate regarding the reclassification of psychedelics, LSD remains a Schedule I controlled substance in the United States, denoting no currently accepted medical use and a high potential for abuse.[10] Its psychological effects include euphoria; perceptual distortions such as illusions, pseudohallucinations, and synesthesia; and disturbances in cognition and time perception.[21] Some individuals may develop persistent sequelae, including mood instability and hallucinogen persisting perception disorder.[22] Reported neurocognitive impairments include deficits in coordination, reaction time, and memory.[23]

Although some studies have shown optimism regarding LSD's therapeutic potential,[24] recognition of its possible severe neuropsychiatric side effects remains essential. As interest in psychedelic-assisted interventions reemerges in psychiatric research, this case underscores the importance of vigilance regarding acute and chronic adverse effects. For primary care and frontline clinicians, particularly those in pediatric and adolescent care, early recognition of catatonic features and a thorough assessment of recent substance use are critical. Early suspicion can expedite psychiatric consultation, initiation of benzodiazepine trials, and transfer to higher levels of care when indicated. This case further reinforces the need for primary care providers to document detailed substance use histories, remain alert to uncommon presentations such as LSD-related catatonia, and coordinate multidisciplinary care to improve outcomes in similar clinical scenarios.

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