

Acute Hallucinogen Intoxication Following Ingestion of a Commercially Available Mushroom Extract Product: A Case Report

Review began 02/19/2026

Review ended 06/10/2026

Published 06/13/2026

© Copyright 2026

Udoro et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.110791

Akemi J. Udoro¹, Yadanar Yukyi^{2, 1}, Ali Z. Ansari^{3, 1}, Hiram A. Gandara^{4, 1}, Fizza Ahmed^{5, 1}, Derek Hunt¹

1. Department of Emergency Medicine, Merit Health Wesley, Hattiesburg, USA 2. Department of Psychiatry, William Carey University College of Osteopathic Medicine, Hattiesburg, USA 3. Department of Family Medicine, Prisma Health Tuomey Hospital, Sumter, USA 4. Department of Internal Medicine, HCA Florida Largo Hospital, Largo, USA 5. Department of Neurology, Lake Erie College of Osteopathic Medicine, Bradenton, USA

Corresponding author: Ali Z. Ansari, dr.ansari.do@gmail.com

Abstract

Psilocybin is a naturally occurring hallucinogen known to produce alterations in perception, cognition, and autonomic function through its effects on central serotonergic pathways. Products marketed as “mushroom” formulations may contain psilocybin or related psychoactive compounds despite ambiguous labeling and limited regulatory clarity, creating diagnostic challenges for clinicians in acute care settings. We present the case of an 18-year-old female with no prior medical or psychiatric history who presented to the emergency department with sudden-onset severe anxiety, agitation, tremors, diaphoresis, and vivid visual and auditory hallucinations shortly after ingesting a commercially available mushroom product purchased from a gas station. According to the patient and her boyfriend, she rapidly developed paranoid ideation and marked perceptual distortions, including the belief that her surroundings were moving and threatening her, prompting emergent evaluation. On arrival, she was tachycardic and tachypneic with dilated pupils, pronounced psychomotor agitation, and hypervigilant behavior, while remaining alert, oriented, and without focal neurologic deficits. Initial diagnostic evaluation revealed mild metabolic abnormalities without evidence of co-ingestion, pregnancy, or end-organ dysfunction, and electrocardiography demonstrated an apparent irregular rhythm initially concerning for atrial fibrillation that was subsequently determined to represent sinus rhythm with tremor-related artifact. The diagnosis of psilocybin-associated hallucinogen intoxication was reached through careful history-taking, exclusion of alternative etiologies such as primary psychosis, serotonin syndrome, or other toxic ingestions, and observation of symptom resolution with supportive care. Diagnostic uncertainty was heightened by the absence of confirmatory toxicology testing and the nonspecific labeling of the ingested product, necessitating reliance on clinical pattern recognition and response to treatment. The patient was managed with intravenous fluids and repeated doses of benzodiazepines, resulting in gradual resolution of autonomic instability, agitation, and hallucinations, and she returned to baseline mental status prior to discharge. This case highlights the diagnostic challenges posed by commercially available mushroom products and emphasizes the importance of comprehensive clinical evaluation and supportive management in suspected psilocybin intoxication. Recognition of characteristic clinical features, coupled with exclusion of dangerous mimics, remains essential for safe disposition and effective patient counseling.

Categories: Psychiatry, Emergency Medicine, Substance Use and Addiction

Keywords: acute psychosis, agitation, auditory hallucinations, autonomic instability, hyperadrenergic state, mushroom ingestion, psilocybin, retail-purchased substances, substance-related disorders, visual hallucinations

Introduction

Psilocybin is a naturally occurring hallucinogenic compound found in approximately 200 species of mushrooms, commonly referred to as “magic mushrooms” [1,2]. It has a long history of use in ritualistic and spiritual practices, as well as recreational use, and more recently has been explored in controlled research settings for potential therapeutic applications, including treatment of depression, anxiety, and post-traumatic stress disorder [3]. Upon ingestion, psilocybin is rapidly metabolized to its active form, psilocin, which acts primarily as a partial agonist at serotonin 5-HT_{2A} receptors in the central nervous system [2]. This serotonergic activity produces profound alterations in sensory perception, cognition, and emotional processing, which may manifest as vivid visual and auditory hallucinations, synesthesia, time distortion, and marked changes in mood or affect [4]. In addition to these neuropsychiatric effects, psilocybin can also induce autonomic changes, including transient elevations in heart rate and blood pressure, pupillary dilation, tremor, diaphoresis, and hyperthermia, reflecting a hyperadrenergic state [5]. While psilocybin has historically been ingested in its natural mushroom form, it is increasingly found in commercially available formulations, including powders, pills, capsules, and vape shop products [6]. These commercial preparations often lack accurate labeling and may contain highly variable concentrations of active compounds or other adulterants, leading to unpredictable and sometimes severe pharmacologic effects, which can complicate both clinical assessment and management [7]. Their increasing availability in gas stations, vape shops,

How to cite this article

Udoro A J, Yukyi Y, Ansari A Z, et al. (June 13, 2026) Acute Hallucinogen Intoxication Following Ingestion of a Commercially Available Mushroom Extract Product: A Case Report. Cureus 18(6): e110791. DOI 10.7759/cureus.110791

smoke shops, and online marketplaces has expanded public access to psychoactive mushroom-derived products that are often marketed as legal wellness or dietary supplements despite uncertain composition and limited regulatory oversight [6,7]. As a result, emergency clinicians are increasingly likely to encounter patients presenting with intoxication after ingestion of products whose active constituents and potency cannot be readily determined. Because routine hospital toxicology screens do not typically detect psilocybin or many related hallucinogenic compounds, clinicians frequently rely on recognition of characteristic toxidromes, careful history-taking, and exclusion of alternative etiologies when evaluating suspected hallucinogen intoxication. This case highlights the diagnostic and management challenges encountered in the emergency department following ingestion of a commercially available mushroom extract product with uncertain composition, as illustrated by the present presentation, and emphasizes the importance of careful clinical assessment in similar cases of suspected hallucinogen intoxication.

Case Presentation

An 18-year-old female with no known past medical or psychiatric history presented to the emergency department accompanied by her boyfriend for the evaluation of acute-onset neuropsychiatric and autonomic symptoms following ingestion of a commercially available “mushroom extract” product purchased from a local gas station. At baseline, the patient was described as high-functioning, with no history of anxiety, mood disorders, psychosis, attention-deficit disorder, seizure disorder, or prior emergency department visits for behavioral or substance-related concerns. She was not taking any prescription or over-the-counter medications and denied the use of herbal supplements. According to both the patient and her boyfriend, she had no prior exposure to hallucinogenic substances, including psilocybin-containing mushrooms, lysergic acid diethylamide (LSD), or other serotonergic agents, and reported no history of recreational drug use, including cannabis or stimulant products.

The ingested product had been purchased legally from a nontraditional retail setting and was marketed as a mushroom-derived extract with packaging that suggested a plant-based or “natural” formulation. The labeling did not clearly identify specific active psychoactive ingredients, standardized concentrations, or regulatory approval status. The patient reported that the product was displayed alongside other novelty or wellness items and was not presented as an illicit substance. She and her boyfriend stated that the product’s legal availability and mushroom-based branding contributed to her perception that it was low-risk and safe for recreational experimentation. She denied any intention of self-harm and stated that ingestion was motivated by curiosity rather than emotional distress or peer pressure.

Further history revealed that approximately one hour prior to arrival, while at home, the patient ingested multiple “mushroom extract pills” sold under the brand name Silly Dots. She stated that she consumed the pills in a single episode rather than spacing them over time. Although she was unable to provide an exact dose, she believed the total amount consumed was either 1,200 mg or 1,800 mg based on the number of capsules taken and the milligram strength listed on the product label. She acknowledged that she did not fully understand whether the labeled dose referred to total mushroom extract, a proprietary blend, or an active psychoactive compound. She also reported that she did not research the product prior to ingestion and was unaware of the expected onset time, potency, or potential adverse effects.

Within approximately 20 to 30 minutes of ingestion, the patient began experiencing heightened sensory awareness and mild restlessness, which quickly progressed to intense anxiety and a sense of impending doom. Over the next half hour, her symptoms escalated substantially, with the development of pronounced psychomotor agitation, generalized tremulousness, intermittent involuntary muscle contractions, diaphoresis, and a strong subjective sensation of internal heat despite the absence of documented fever. She reported that her heart was “racing” and described difficulty controlling her breathing as panic intensified. Concurrently, she developed vivid perceptual disturbances, including complex visual hallucinations of shadow-like figures, distortions in spatial perception in which walls appeared to bend and move inward, and auditory hallucinations consisting of indistinct voices and exaggerated environmental sounds. These experiences were accompanied by escalating paranoia, marked hypervigilance, and repeated verbalizations that something catastrophic was happening to her body and mind. She described a distressing loss of control over her thoughts and surroundings, stating that reality felt “fragmented” and “unrecognizable.” Her boyfriend observed behavior acutely inconsistent with her baseline personality, including pacing, intermittently freezing in place, clutching him for reassurance, withdrawing in fear, and crying. Despite severe psychological distress, he denied any seizure-like activity, tonic-clonic movements, loss of consciousness, trauma, urinary or fecal incontinence, tongue biting, or postictal confusion. The rapid progression and intensity of these symptoms prompted him to transport her to the emergency department for evaluation.

The patient consistently denied co-ingestion of alcohol, cannabis, prescription medications, over-the-counter agents, energy drinks, or illicit substances. She denied the use of serotonergic medications such as selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, or stimulant agents that might predispose to serotonin toxicity. She further denied suicidal ideation, self-harm intent, or ingestion with the purpose of achieving intoxication beyond recreational curiosity. There was no prior history of panic attacks, manic symptoms, psychosis, dissociation, or prior emergency psychiatric evaluation. She denied chest pain, dyspnea, headache, visual loss, focal weakness, nausea, vomiting, abdominal pain, or prior similar episodes.

There was no personal history of mood disorders, psychotic disorders, seizure disorder, cardiovascular disease, or substance use disorder. Family history was noncontributory, with no known psychiatric illnesses, epilepsy, sudden cardiac death, or heritable neurologic conditions among first-degree relatives. Social history was limited but did not reveal ongoing substance use, recent psychosocial stressors, sleep deprivation, dietary changes, travel, toxin exposure, or recent infectious symptoms.

Upon arrival to the emergency department, the patient appeared markedly anxious and visibly distressed. She was seated upright on the stretcher, intermittently tremulous, with restless movements of her upper extremities and frequent shifting of position. Psychomotor agitation was evident, though she remained cooperative with staff and was redirectable with calm verbal reassurance. She demonstrated exaggerated startle responses to routine environmental stimuli, including monitor alarms and hallway noises, consistent with heightened sensory sensitivity. Initial vital signs revealed tachycardia and tachypnea, while blood pressure, oral temperature, and oxygen saturation remained within acceptable physiologic limits. She was afebrile. Her skin was warm and dry without flushing, urticaria, rash, track marks, or cyanosis. There was no evidence of diaphoresis severe enough to suggest hyperthermia. Head and neck examination was unremarkable, with no signs of trauma, meningismus, or thyromegaly. Pupils were dilated to approximately 8 mm bilaterally but were symmetric and briskly reactive to light. Extraocular movements were intact without nystagmus, and conjunctivae were clear. Oral mucosa was moist, with no xerostomia, tongue fasciculations, or evidence of tongue biting.

Cardiovascular examination revealed tachycardia with a regular rhythm on auscultation and no appreciable murmurs, rubs, or gallops. Peripheral pulses were symmetric and strong, and capillary refill was less than two seconds. No peripheral edema was present. Pulmonary examination demonstrated clear breath sounds bilaterally with good air movement and no wheezes, crackles, stridor, or accessory muscle use. Respiratory effort was mildly increased in rate but not labored. Abdominal examination was benign, with a soft, non-tender, and non-distended abdomen, normal bowel sounds, and no hepatosplenomegaly. Musculoskeletal examination showed a full range of motion in all extremities without rigidity, lead-pipe stiffness, hypertonia, or inducible or spontaneous clonus. No hyperreflexia was observed. Neurologically, the patient was alert and fully oriented to person, place, time, and situation. Cranial nerves II–XII were intact. Motor strength was 5/5 throughout, sensation to light touch was preserved, and coordination was grossly normal. There were no focal neurologic deficits, ataxia, or dysmetria. Speech was fluent and coherent but somewhat pressured, reflecting heightened anxiety. Psychiatric examination was notable for severe anxiety, paranoid ideation, perceptual disturbances, and impaired judgment attributable to acute intoxication. Insight was limited during the peak of symptoms but improved with reassurance. She endorsed ongoing visual and auditory hallucinations during initial assessment but retained partial awareness that these experiences might be substance-induced.

Given the acuity and severity of presentation, a comprehensive diagnostic evaluation was undertaken to assess for metabolic abnormalities, toxicologic exposures, structural or functional neurologic pathology, and infectious etiologies. Laboratory studies were obtained to evaluate for electrolyte disturbances, hypoglycemia, renal or hepatic dysfunction, and potential co-ingestions. Initial results demonstrated leukocytosis with neutrophil predominance, mild hypokalemia, and a low serum bicarbonate level. These findings were interpreted as likely reflective of acute physiologic stress, hyperventilation, and sympathetic activation rather than primary infection or systemic inflammatory pathology. Renal and hepatic function tests were within normal limits, arguing against end-organ injury. Serum acetaminophen and salicylate levels were undetectable, reducing concern for common toxic co-ingestions. Urine pregnancy testing was negative. A summary of laboratory findings is presented in Table 1.

Laboratory Test	Result	Reference Range
White blood cell count	13.7 × 10 ³ /μL	4.0–10.5 × 10 ³ /μL
Hemoglobin	12.7 g/dL	12.0–16.0 g/dL
Hematocrit	37.4%	36–46%
Platelets	276 × 10 ³ /μL	150–400 × 10 ³ /μL
Sodium	137 mmol/L	135–145 mmol/L
Potassium	3.2 mmol/L	3.5–5.1 mmol/L
Chloride	107 mmol/L	98–107 mmol/L
Bicarbonate	19 mmol/L	22–29 mmol/L
BUN	5 mg/dL	7–20 mg/dL
Creatinine	0.66 mg/dL	0.6–1.3 mg/dL
Glucose	108 mg/dL	70–99 mg/dL
Calcium	9.6 mg/dL	8.5–10.5 mg/dL
AST	16 U/L	10–40 U/L
ALT	<7 U/L	7–56 U/L
Total bilirubin	0.50 mg/dL	0.2–1.2 mg/dL
Acetaminophen level	<2.0 μg/mL	10–30 μg/mL
Salicylate level	<3.0 mg/dL	15–30 mg/dL
Urine pregnancy test	Negative	Negative

TABLE 1: Initial laboratory evaluation demonstrating mild leukocytosis with neutrophil predominance, hypokalemia, and low serum bicarbonate in the setting of acute hallucinogen intoxication, with otherwise unremarkable renal and hepatic function and no evidence of common toxic co-ingestions.

BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase

A 12-lead electrocardiogram (ECG) was obtained to evaluate for arrhythmia, conduction abnormalities, or QT interval prolongation in the context of sympathomimetic features. The initial automated interpretation suggested an irregular rhythm concerning atrial fibrillation, with a ventricular rate of 86 beats per minute, QRS duration of 81 ms, QT interval of 358 ms, and corrected QT interval (QTc) of 429 ms (Figure 1). However, bedside clinical correlation revealed visible generalized tremors at the time of acquisition, and repeat review demonstrated consistent P waves preceding each QRS complex. The irregular baseline was ultimately attributed to motion artifact secondary to tremulousness rather than true atrial fibrillation. No ischemic changes, pathologic ST-segment deviations, or clinically significant QT prolongation were identified.

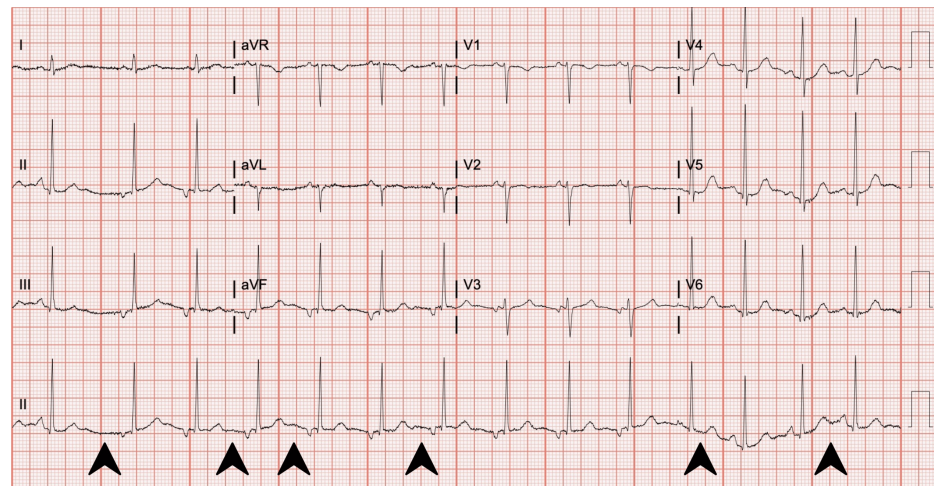


FIGURE 1: Twelve-lead electrocardiogram demonstrating sinus rhythm with significant motion artifact secondary to generalized tremors (selected artifacts are indicated by black arrowheads in lead II).

The patient was placed on continuous cardiac telemetry and pulse oximetry monitoring upon arrival to the emergency department, given the presence of tachycardia, autonomic activation, and initial ECG uncertainty. She was assigned to a monitored treatment bay with reduced environmental stimuli to minimize sensory overload and exacerbation of perceptual disturbances. A supportive care approach was implemented, consistent with standard management of suspected hallucinogen intoxication. Intravenous (IV) access was established, and isotonic crystalloid fluid (lactated Ringer's solution) was administered to address potential dehydration, compensate for insensible losses related to diaphoresis and tachypnea, and mitigate sympathetic overactivity. Environmental modifications, including dimmed lighting and minimized auditory stimulation, were employed to reduce anxiety and perceptual amplification.

Despite initial supportive measures, including environmental modification and initiation of IV fluids, the patient continued to demonstrate marked psychomotor agitation, generalized tremulousness, persistent tachycardia, and ongoing distressing visual and auditory hallucinations during the early phase of her emergency department course. She remained fearful, intermittently tearful, and described an intense sense of internal restlessness and loss of control. Approximately one hour after arrival, given persistent agitation and escalating anxiety that interfered with redirection and clinical assessment, she was administered 2 mg of IV lorazepam for acute anxiolysis and sedation. This initial dose was intended to reduce severe anxiety, attenuate sympathetic overactivity contributing to tachycardia and tremor, and mitigate neuromuscular excitation. Although partial improvement was observed, including mild reduction in pacing and verbal distress, she continued to experience tremulousness and perceptual disturbances. A second 2 mg IV dose was administered approximately 20 to 30 minutes later due to ongoing agitation and hallucinations. Over the subsequent period of observation, an additional 2 mg dose was given as needed for recurrent restlessness and persistent tremor, with dosing titrated to clinical response. Benzodiazepine therapy was selected as first-line pharmacologic management given its established role in treating agitation and autonomic hyperactivity associated with hallucinogen intoxication, while avoiding dopamine antagonism that could complicate serotonergic states. Following sequential dosing, the patient demonstrated progressive improvement: motor restlessness diminished, tremors decreased in amplitude and frequency, heart rate trended downward, and the intensity of visual and auditory hallucinations lessened. Her speech became less pressured, affect stabilized, and she was increasingly able to participate in coherent, linear conversation and engage meaningfully with staff.

Throughout her emergency department course, the patient underwent serial reassessments to monitor for progression of symptoms or development of complications. Particular attention was given to features that might suggest serotonin toxicity, including hyperthermia, sustained autonomic instability, inducible or spontaneous clonus, hyperreflexia, muscular rigidity, or worsening alteration in mental status. None of these findings emerged. Her temperature remained within normal limits on repeated measurements, blood pressure was stable without labile hypertensive episodes, and neurologic examinations demonstrated no hypertonia, clonus, focal deficits, or deterioration in cognition. She maintained airway protection and hemodynamic stability throughout observation. Given the reported timing of ingestion - approximately one hour prior to arrival - and her steady clinical improvement with supportive care and benzodiazepine therapy, gastrointestinal decontamination with activated charcoal was not indicated. There was no evidence of co-ingestion or persistent instability warranting toxicology consultation, advanced airway intervention, or intensive care admission. In the absence of confirmatory toxicologic testing, the diagnosis remained clinical; however, the clear temporal association with ingestion of a mushroom-derived commercial product, the

presence of mydriasis, tachycardia, anxiety, tremulousness, and prominent perceptual disturbances, along with predictable improvement over several hours, was most consistent with acute hallucinogen intoxication, presumed secondary to psilocybin or a psilocybin-like serotonergic compound.

Over several hours of monitored observation, the patient demonstrated steady clinical recovery. Heart rate and respiratory rate gradually normalized. Tremulousness resolved, and diaphoresis subsided. She no longer endorsed visual or auditory hallucinations and denied ongoing perceptual distortion. On repeat psychiatric evaluation, she was calm, cooperative, and able to participate meaningfully in discussion regarding her care. Thought processes were linear and goal-directed. She demonstrated intact orientation, memory, and attention. Insight improved substantially as intoxication resolved; she acknowledged that her symptoms were likely related to ingestion of the mushroom product and expressed embarrassment and regret regarding the decision to consume it. She denied suicidal ideation, homicidal ideation, or persistent paranoia. Decision-making capacity was assessed and deemed intact. After shared decision-making involving the patient, her boyfriend, and family members who had arrived during her observation period, she was considered medically stable for discharge with reliable supervision at home.

Prior to discharge, the patient received detailed counseling regarding the risks associated with unregulated and ambiguously labeled psychoactive products, particularly those marketed in vape shops, gas stations, and similar retail environments. Education focused on the unpredictability of dose, potential adulterants, variability in active compound concentration, and the risk of severe psychological or physiologic reactions even in individuals without prior psychiatric vulnerability. She was advised to abstain from future use of mushroom-derived or other psychoactive substances and was provided with guidance on recognizing symptoms of substance toxicity. Harm-reduction principles were discussed in a nonjudgmental manner. Written discharge instructions included strict return precautions for recurrent hallucinations, persistent anxiety, chest pain, hyperthermia, confusion, or any new neurologic symptoms. She was advised to follow up with her primary care provider within several days for reassessment.

At the outpatient follow-up with her primary care provider several days later, the patient reported complete resolution of symptoms. She denied recurrence of hallucinations, anxiety, tremors, insomnia, mood lability, or autonomic complaints. She had returned to her usual daily activities, including school and social engagement, without difficulty. She denied flashbacks, persistent perceptual disturbances, or symptoms suggestive of hallucinogen persisting perception disorder. She also denied any subsequent substance use. Mental status at follow-up was normal, and no additional medical or psychiatric intervention was deemed necessary. She was reassured regarding the absence of apparent long-term sequelae and was again counseled regarding avoidance of unregulated psychoactive products. At the time of follow-up, she had returned fully to her baseline functional and psychological status. Overall, this presentation was characterized by an acute onset of anxiety, psychomotor agitation, autonomic activation (tachycardia, mydriasis, diaphoresis), tremor, and prominent visual and auditory hallucinations with preserved orientation and cognition, a constellation of findings consistent with a hallucinogen toxidrome.

Discussion

A central and particularly concerning feature of this case is the ingestion of a commercially packaged product marketed broadly as a “mushroom extract,” purchased from a gas station and sold under a brand name that did not clearly disclose its specific psychoactive constituents or their concentrations. Unlike naturally occurring psilocybin-containing mushrooms, where approximate potency and expected clinical effects are at least described in existing literature, retail “mushroom” formulations exist within a largely unregulated marketplace [8]. These products may contain variable amounts of psilocybin, psilocybin analogs, synthetic tryptamines, or other serotonergic compounds and, in some instances, may include entirely different psychoactive substances not traditionally associated with naturally occurring fungi [6]. Labeling frequently emphasizes vague terms such as “proprietary blend” or “extract,” without transparent ingredient lists, quantitative dosing information, or standardized potency metrics. As a result, consumers may mistakenly perceive these products as benign, legal, or comparable to non-psychoactive dietary supplements, when, in fact, their pharmacologic effects may be unpredictable and potentially severe.

In emergency medicine, presentations such as this are most effectively interpreted through a toxidrome-based framework, which enables rapid diagnostic narrowing when confirmatory toxicologic testing is unavailable. In this case, the patient was unable to verify the exact substance ingested, the dose consumed, or the presence of additional active compounds. No confirmatory toxicologic testing was available to identify specific serotonergic agents, and routine hospital toxicology screens do not typically detect psilocybin or many synthetic tryptamines. Consequently, the diagnosis relied heavily on clinical pattern recognition, temporal association with ingestion, and systematic exclusion of alternative etiologies. The constellation of acute-onset perceptual disturbances, autonomic activation, mydriasis, anxiety, and favorable response to benzodiazepines supported a hallucinogen-induced process; however, the inability to definitively characterize the ingested compound represents a meaningful diagnostic limitation [9]. This uncertainty has important implications for emergency clinicians, as the increasing availability of ambiguously labeled psychoactive products in nontraditional retail environments complicates risk assessment, counseling, and anticipatory management [7]. A degree of diagnostic uncertainty remains inherent in this case due to the absence of confirmatory toxicologic testing and the inability to verify the

exact composition of the ingested product, necessitating reliance on clinical presentation, temporal association, and exclusion of alternative etiologies.

The patient's presentation was consistent with psilocybin-associated hallucinogen intoxication, based on rapid onset of neuropsychiatric and autonomic symptoms within 30 to 60 minutes of ingesting a commercially packaged mushroom-derived product, including intense anxiety, psychomotor agitation, mydriasis, tremor, diaphoresis, tachycardia, and complex visual and auditory hallucinations [2,4,6]. She remained fully oriented, and serial neurologic examinations showed no focal deficits or cognitive decline. The diagnosis was established through careful history-taking, clear temporal association with ingestion, exclusion of primary psychosis, serotonin syndrome, and other toxic ingestions, and observation of gradual improvement with supportive care and benzodiazepine therapy. Although autonomic activation and tremor were present, she did not develop sustained hyperthermia, clonus, hyperreflexia, muscular rigidity, labile blood pressure, or altered mental status. Her hemodynamics remained stable aside from sinus tachycardia, and no advanced interventions, cyproheptadine, or intensive care monitoring were required. The absence of persistent psychosis or delirium after symptom resolution further supported a self-limited, substance-induced process, reinforcing that her clinical course represented acute psilocybin-associated hallucinogen intoxication.

Another important aspect of this case was the initial ECG interpretation suggesting atrial fibrillation, which directly impacted clinical decision-making. Automated analysis reported an irregular rhythm, raising concern for a potentially serious cardiac arrhythmia in a patient already experiencing sympathetic overactivity from hallucinogen intoxication. However, bedside assessment revealed pronounced tremulousness due to psychomotor agitation at the time of acquisition. Careful review of the ECG waveform demonstrated organized atrial activity with consistent P waves preceding QRS complexes, confirming sinus rhythm and identifying the irregular baseline as motion artifact. Recognizing this artifact prevented unnecessary cardiology consultation, additional interventions, or potential hospital admission, allowing the medical team to focus on appropriate supportive care and benzodiazepine management [10]. This observation highlights a practical implication of the case: in patients with acute hallucinogen intoxication, visible motor activity can significantly interfere with automated diagnostic tools, and reliance on these readings without clinical correlation can lead to misinterpretation and overtreatment. In this patient, careful correlation of ECG findings with her physical presentation ensured accurate diagnosis, avoided unnecessary escalation of care, and emphasized the importance of integrating clinical context into the interpretation of diagnostic studies during acute toxicologic events.

Management in this case closely reflected established principles for treating acute hallucinogen intoxication and was directly guided by the patient's clinical presentation. Supportive care measures, including placement in a low-stimulation environment, continuous monitoring, reassurance, and titrated IV lorazepam, resulted in gradual but consistent improvement in anxiety, psychomotor agitation, tremors, and perceptual disturbances [11]. Lorazepam proved highly effective, providing rapid anxiolysis, reduction in sympathetic overactivity, and attenuation of neuromuscular excitation, which allowed the patient to regain a sense of control and engage in coherent conversation. Multiple sequential doses were required, each titrated to clinical effect, highlighting the importance of individualized dosing in the management of acute hallucinogen intoxication. Its use obviated the need for antipsychotics, which could worsen serotonergic activity, as well as physical restraints or invasive interventions. The patient remained hemodynamically stable, did not require airway support, active cooling, vasopressors, or intensive care monitoring, and demonstrated predictable improvement with this targeted pharmacologic approach. Clinically, this case highlights the utility of benzodiazepines as first-line therapy in similar presentations and provides a practical framework for other physicians encountering acute hallucinogen toxicity. Recognizing that supportive care combined with judicious pharmacologic intervention can safely and effectively manage severe agitation and autonomic activation may help clinicians avoid unnecessary escalation, reduce risk of iatrogenic complications, and differentiate isolated hallucinogen intoxication from more severe toxicologic or emergent psychiatric conditions. The predictable response observed in this patient reinforces the importance of structured bedside assessment, continuous monitoring, and individualized treatment strategies in guiding safe disposition and optimizing patient outcomes.

Conclusions

This case describes acute hallucinogen intoxication in an otherwise healthy 18-year-old female following ingestion of a commercially available, ambiguously labeled "mushroom extract" product purchased from a gas station. The presentation was characterized by rapid-onset perceptual disturbances, autonomic activation, and severe anxiety, with diagnostic uncertainty compounded by the absence of confirmatory toxicologic testing and an initially misleading electrocardiographic interpretation due to motion artifact. Careful history-taking, serial clinical reassessment, exclusion of dangerous mimics such as serotonin syndrome and primary psychosis, and correlation of diagnostic studies with bedside findings were essential in establishing a working diagnosis. Overall, the clinical presentation is consistent with a hallucinogen toxidrome, and recognition of this pattern, even in the absence of confirmatory toxicologic testing, supported timely diagnosis and management. The patient's complete recovery with supportive care and benzodiazepine therapy in this case is consistent with the typically self-limited clinical course described in isolated hallucinogen intoxication in otherwise stable patients. As commercially available mushroom-

derived products become increasingly accessible, clinicians should remain vigilant for similar presentations and recognize that legal retail availability does not equate to predictable composition or safety.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ali Z. Ansari, Akemini J. Udoro, Fizza Ahmed, Yadanar Yukyi, Hiram A. Gandara

Acquisition, analysis, or interpretation of data: Ali Z. Ansari, Akemini J. Udoro, Derek Hunt, Yadanar Yukyi

Drafting of the manuscript: Ali Z. Ansari, Akemini J. Udoro, Fizza Ahmed, Yadanar Yukyi, Hiram A. Gandara

Critical review of the manuscript for important intellectual content: Ali Z. Ansari, Akemini J. Udoro, Derek Hunt, Yadanar Yukyi

Supervision: Derek Hunt

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Plotnik L, Gibbs G, Graham T: Psilocybin conspectus: status, production methods, and considerations. *Int J Med Mushrooms*. 2022, 24:1-11. [10.1615/IntJMedMushrooms.2021041921](https://doi.org/10.1615/IntJMedMushrooms.2021041921)
2. Kargbo RB: Psilocybin therapeutic research: the present and future paradigm. *ACS Med Chem Lett*. 2020, 11:399-402. [10.1021/acsmchemlett.0c00048](https://doi.org/10.1021/acsmchemlett.0c00048)
3. Bienemann B, Ruschel NS, Campos ML, Negreiros MA, Mograbi DC: Self-reported negative outcomes of psilocybin users: a quantitative textual analysis. *PLoS One*. 2020, 15:e0229067. [10.1371/journal.pone.0229067](https://doi.org/10.1371/journal.pone.0229067)
4. Smigielski L, Kometer M, Scheidegger M, Krähenmann R, Huber T, Vollenweider FX: Characterization and prediction of acute and sustained response to psychedelic psilocybin in a mindfulness group retreat. *Sci Rep*. 2019, 9:14914. [10.1038/s41598-019-50612-3](https://doi.org/10.1038/s41598-019-50612-3)
5. Ley L, Holze F, Arikci D, et al.: Comparative acute effects of mescaline, lysergic acid diethylamide, and psilocybin in a randomized, double-blind, placebo-controlled cross-over study in healthy participants. *Neuropsychopharmacology*. 2023, 48:1659-67. [10.1038/s41386-023-01607-2](https://doi.org/10.1038/s41386-023-01607-2)
6. Correia MS, Gonzaga MJ, Temple C, Gerona RR: Quantitative analysis of recreational psychoactive mushroom gummies in Portland, Oregon. *Clin Toxicol (Phila)*. 2025, 63:261-6. [10.1080/15563650.2025.2450240](https://doi.org/10.1080/15563650.2025.2450240)
7. Reinert JP, Colunga K, Etuk A, Richardson V, Dunn RL: Management of overdoses of salvia, kratom, and psilocybin mushrooms: a literature review. *Expert Rev Clin Pharmacol*. 2020, 13:847-56. [10.1080/17512433.2020.1794811](https://doi.org/10.1080/17512433.2020.1794811)
8. Gibbons WJ Jr, McKinney MG, O'Dell PJ, Bollinger BA, Jones JA: Homebrewed psilocybin: can new routes for pharmaceutical psilocybin production enable recreational use?. *Bioengineered*. 2021, 12:8863-71. [10.1080/21655979.2021.1987090](https://doi.org/10.1080/21655979.2021.1987090)
9. Caplan RA, Zuflacht JP, Barash JA, Fehnel CR: Neurotoxicology syndromes associated with drugs of abuse. *Neurol Clin*. 2020, 38:983-96. [10.1016/j.ncl.2020.08.005](https://doi.org/10.1016/j.ncl.2020.08.005)
10. Pérez-Riera AR, Barbosa-Barros R, Daminello-Raimundo R, de Abreu LC: Main artifacts in electrocardiography. *Ann Noninvasive Electrocardiol*. 2018, 23:e12494. [10.1111/anec.12494](https://doi.org/10.1111/anec.12494)
11. Barnett BS, Koons CJ, Van den Eynde V, Gillman PK, Bodkin JA: Hypertensive emergency secondary to combining psilocybin mushrooms, extended release dextroamphetamine-amphetamine, and tranlylcypromine. *J Psychoactive Drugs*. 2025, 57:297-303. [10.1080/02791072.2024.2368617](https://doi.org/10.1080/02791072.2024.2368617)