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Case Report

Inhalant-Induced Psychotic Disorder: A Case Report

Vundavalli Jaya Harikha[1], Sangha Mitra Godi[1*], Vijaya Chandra Reddy Avula[1], Sree Pruthvi Mithra Raman[1]

[1]Department of Psychiatry, All India Institute of Medical Sciences (AIIMS),Andhra Pradesh Mangalagiri, India

***Corresponding Author:** Sangha Mitra Godi, Email: mitratriam@gmail.com

Abstract

Background: Inhalants are volatile organic compounds (VOCs) abused to achieve psychostimulant effects. VOCs are found in substances such as paint thinners, glues, and adhesives.

Case Presentation: This case illustrates a 27-year-old unmarried, unemployed man from rural Andhra Pradesh with a three-year history of volatile inhalant use, specifically Fevicol. His inhalant use began during college and escalated from occasional use to daily consumption of up to 1 Liter per day through huffing and bagging. He reported a feeling of euphoria and occasional drowsiness accompanied by redness of the eyes. Parallel to the increase in his inhalant use, noticeable behavioral changes, including decreased social interaction, self-talking, irritability, frequent disputes with family members, and multiple job changes, were observed. Upon interview, psychotic symptoms including auditory hallucinations and persecutory delusions, started around one year ago, prompting inpatient psychiatric admission. A diagnosis of volatile inhalant-induced psychotic disorder (ICD-11) was made. Management relied on antipsychotics (risperidone) as the cornerstone, along with cognitive behavioral therapy, motivational enhancement therapy and relapse prevention for sustained recovery.

Discussion: This case highlights distinctive aspects, including prolonged duration of psychotic symptoms following chronic inhalant exposure, as well as the aggravation of psychotic symptoms (mainly auditory hallucination) occurring 15–20 minutes after acute inhalant use. The nicotine self-medication hypothesis has been proposed to explain this phenomenon, suggesting that nicotine may alleviate distress during acute use.

Conclusion: The focus was not only to explain the psychiatric sequelae of inhalant abuse but also to underscore the importance of comprehensive management strategies for adequate recovery and to unveil the roles of gamma-aminobutyric acid (GABA) disinhibition, glutamate, and dopamine dysregulation. Ultimately, the combination of pharmacotherapy and behavioral interventions is essential. To decrease experimentation with inhalants, regulation alone is insufficient without education and awareness-raising. A specific combination of strategies at three different levels, the government (consistent stringent rules at state and national levels, restriction of sales to minors, and community and school-based interventions), producers (adding deterrents and replacing harmful chemicals with safer alternatives), and consumers (education on health consequences and legal implications), can be implemented in India.

Keywords: Inhalant abuse, Glue abuse, Volatile organic compounds, Auditory hallucination, Gamma-aminobutyric acid

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Introduction

Inhalant abuse in Western countries has been documented since the19th century, while inhalant use among the Indian population has been reported only since the 1970s.[1] In a 2019 survey, the National Drug Dependence Treatment Centre (NDDTC), AIIMS, New Delhi, reported that 458,000 children and 1.8 million adults in India need help for problematic inhalant use, with Uttar Pradesh, Madhya Pradesh, Maharashtra, Delhi, and Haryana being highly affected.[2]

Common categories of inhalants include volatile solvents, aerosols, gases, and nitrites, classified based on the forms in which they are commonly found in household, industrial, and medical products.

Volatile organic compounds(VOCs) or solvents are found in substances such as paint thinners, paint removers, dry cleaning fluids, glues, typewriter correction fluids, gasoline, adhesives, varnishes, dry cleaning agents, deodorants, hair sprays, etc.[3] Commercially sold products most frequently used for inhalation include solvents (34.9%), followed by glues and adhesives (22.9%).[4] Among the various inhalant abuse methods, huffing and bagging were the most employed methods, according to previous studies.[5]

Fevicol (Fevicol SR 505), a commonly used adhesive, contains volatile organic compounds (VOCs), including toluene and xylene, which produce chemical vapors that can be harmful when inhaled, producing mind-altering effects. These compounds are absorbed into the bloodstream via three routes, including the lungs, the skin, and the gastrointestinal tract, and metabolized by mixed-function oxidase enzyme systems into methyl hippuric

acid, *o*-cresol for xylene, and benzoic and hippuric acid for toluene before being excreted in the urine.[6] Fevicol is readily available and is often perceived as a harmless household item. However, its misuse can lead to a wide range of neurological and psychological adverse effects. A study by Ramón MF et al which included 109 cases of inhalant use,reported that 72.5% of the individuals were symptomatic, with 62.8% showing central nervous system (CNS) manifestations.[4] Although Fevicol inhalation is relatively rare, prompt recognition and appropriate management are crucial to prevent complications. The prevalence of inhalant use in the adult population is 0.58%, with a higher prevalence of 1.17% observed among children and adolescents.[7] This case study highlights the distinctive aspects of prolonged duration of psychotic symptoms following chronic inhalant exposure, aggravation of auditory hallucination 20 minutes after inhalant abuse, and the nicotine self-medication hypothesis to counteract the psychotic symptoms induced by inhalants, suggesting that inhalants could serve as a new gateway drug.

Case presentation

Here, we report the case of a 27-year-old, unmarried, unemployed male college dropout from rural Andhra Pradesh. He presented to the psychiatry outpatient department with complaints of irritability, aggressive behavior, suspiciousness toward family members and auditory hallucinations. Subsequently, he was admitted to inpatient care for further evaluation and treatment.

On detailed interviewing, the patient reported regular inhalant use and cigarette smoking in a dependent pattern. He reported a history of inhalant (Fevicol SR 505) use for the past three years. He reported starting inhalant use during his college years upon the suggestion of peers, and attributed his inhalant use to its affordability, availability, and feasibility of use. Initially, he used half a 500 mL bottle once a week at gatherings for 2-3 months, and soon increased it to a daily use of one 500 mL Fevicol SR 505 bottle through bagging and huffing methods. This pattern continued for the next six to eight months. He reported feelings of euphoria and pleasure, occasionally accompanied by drowsiness and redness of the eyes following huffing. These effects lasted for one hour and resolved on their own. Over the previous two years, he had increased his usage to two 500 mL Fevicol SR bottles, with a frequency of 10–15 inhalations per day through huffing and bagging.

Symptoms of auditory hallucination, suspiciousness, and other behavioral changes started approximately one year ago. He reported experiencing hearing the voices of family members talking and discussing matters about him, which occurred most of the day and caused distress for him. In addition, there was a noticeable behavior change, characterized by decreased social interaction, irritability, frequent disputes with family members, and multiple changes of jobs, secondary to the hallucinatory experiences. There were reports of observing gum-stained clothes with the smell of Fevicol. He believed that the voices were persistent despite multiple contrary explanations by his family members. The patient reported that, following 10–15 minutes of inhalant use, the voices grew louder and lasted longer, causing severe distress and leading to irritability and aggressive outbursts against family members. The intensity and duration of auditory hallucinations increased during periods of acute inhalant use. This resulted in a pattern of smoking cigarettes following inhalant use to relieve the auditory hallucinations.

The number and frequency of cigarette smoking had increased over the past year to sustain the relief from hallucinatory experiences. This cycle of Fevicol use to achieve euphoria, followed by cigarette smoking to counteract the hallucinatory experiences, persisted. Although attempts were made to refrain from inhalant use, complete cessation was never achieved due to intense cravings and withdrawal symptoms, including irritability, restlessness, sleep disturbance, and fatigue.

General examination revealed a body mass index (BMI) of 30.9 and a weight of 80 kg.Mental status examination revealed delusions of reference and persecution, somatic passivity, and third-person auditory hallucinations. Investigations revealed a urine drug screen positive for prescribed benzodiazepines, hemoglobin level of 9.6 g/dL (anemia can be explained as a complication of chronic inhalant abuse), HbA1c of 6.5%, raised T3 levels (66 ng/dL), normal liver and kidney function, and normal serum electrolytes. Non-contrast CT (NCCT) of the brain showed diffuse thickening of the bilateral ethmoidal sinuses, which were consistent with the MRI brain findings. No other findings were reported from MRI and CT imaging.

He was diagnosed with volatile inhalant-induced psychotic disorder, as per ICD11 code 6C4B.6, and was advised inpatient care. The severity of psychotic symptoms was assessed using the Positive and Negative Syndrome Scale (PANSS) and the Psychotic Symptom Rating Scale: Auditory Hallucination (PSYRATS-AH), revealing baseline scores of 117 and 38, respectively.

As a part of symptom management, the patient was treated with antipsychotic medication (risperidone 6 mg tablet and clonazepam 0.5 mg HS 0-0-1 tablet) (Table 1). His PANSS score decreased following antipsychotic treatment. After two months of treatment with good compliance, the PANSS and PSYRATS scores decreased to 67 and 23, respectively (Table 1).

At a follow-up two months later, the patient remained abstinent from inhalant use and reported no auditory hallucinations, persecutory ideation, or cravings, with adequate socio-occupational functioning. Further maintenance treatment included continuation of

Table 1. Clinical management plan with timeline: pharmacological and non-pharmacological interventions

Time/duration	Pharmacological management	Therapy		Lifestyle modifications	Scales
Baseline	Started on Tab. risperidone 2 mg P/O HS 0-0-1	I. II.	Motivational enhancement therapy Supportive psychotherapy	Activity scheduling chart Dietary modification Physical exercise	PANSS-114 PSYRATS Auditory hallucinations-38
After three weeks, at the time of discharge	On Tab. risperidone 6 mg/day	I. II.	Motivational enhancement therapy Supportive psychotherapy	Activity scheduling chart Dietary modification Physical exercise	PANSS-87 PSYRATS-AH-29
After two months	Maintained on Tab. risperidone 6 mg/day	I. II. III.	Motivational enhancement therapy Relapse prevention therapy Supportive psychotherapy	Activity scheduling chart Dietary modification chart Physical exercise	PANSS-67 PSYRATS Auditory hallucinations-23
Recent follow-up (at 12 months)	Maintained on Tab. risperidone 6 mg/day	• • •	Currently, the patient has no active psychotic symptoms and is pursuing his career. Abstinent from inhalant use and smoking cigarettes (no relapses noticed). The duration since last inhalant use is around 11-12 months, suggesting recovery.		

relapse prevention therapy and a maintenance dose of antipsychotic medication. Cognitive behavioral therapy under supervision was planned for the subsequent follow-ups (Table 1).

Discussion

With the rise in its prevalence each year , inhalant abuse is no longer an iceberg phenomenon, but a growing epidemic, often overshadowed in India due to easy availability, lack of awareness and lack of strict regulations on inhalant purchase. Due to its low cost, ease of accessibility, and rapid mood elevating effects compared to many other psychoactive substances on the market, it is frequently sought after, mostly by people from rural backgrounds, low socio-economic status, poor social support, lower education grades, and those educated up to middle school. Current literature by Kumar S et al and Ismail H et al on the profile of inhalant seekers also supports the factors discussed above.[3,5]

Mental health manifestations, including poor academic performance, criminal and impulsive behavior, low self-esteem, suicidality, and comorbid substance use, are often associated with inhalant use and related disorders.[8] A surge in dopamine and norepinephrine levels in the prefrontal cortex and nucleus accumbens occurring after 30 minutes of vapor exposure[9] can explain the increased intensity and loudness of hallucinations 20–30 minutes following huffing, as demonstrated in the present case. A study by Ismail et alinvolving participants aged 10–19 years found that adhesives were the most predominantly used inhalants. Additionally, 69.5% of primary inhalant users had nicotine use as a major comorbidity.[5,10] The similar observation in the present case report might strengthen the self-medication with nicotine hypothesis. While this hypothesis has been extensively studied in psychiatric disorders such as schizophrenia, with high smoking rates of 80%, there is a lack of evidence supporting its role in inhalant-induced psychotic disorders. Nicotine alleviates both positive and negative symptoms through two mechanisms. First, it acts directly on nicotinic acetylcholine receptors and potentially stabilizes dopamine levels in areas associated with positive symptoms. Second, it exerts an indirect effect by improving cognitive deficits, including working memory and attention, which in turn may indirectly reduce the severity of positive symptoms and improve overall brain functioning.

Fevicol SR is a synthetic rubber adhesive primarily composed of chemicals such as toluene, synthetic resins, polyvinyl compounds, and other volatile solvents. When misused, they are potent enough to cause psychiatric symptoms due to their high volatility, lipophilicity, and probable efficiency in crossing the blood-brain barrier, leading to their storage in cerebral tissues. Although the exact mechanism underlying inhalant-induced psychosis is unclear, evidence suggests several hypotheses involving dopaminergic activation and glutamatergic and gamma-aminobutyric acid GABAergic inhibition, which may play a role in its neuropsychiatric manifestations.[3]

A few case reports have reported the possibility of the onset of psychotic manifestations following consumption of compounds such as toluene, wherein patients presenting with psychosis following toluene use showed improvement in symptoms with abstinence.[6] Another report described a case of five years of continuous occupational exposure to toluene that was associated with irreversible schizophreniform psychosis.[11] Five cases were reported from northern Karnataka, including two cases of depression and one case of psychosis following sniffing petrol and correction fluid, which was treated with risperidone 4 mg/day along with cognitive behavioural therapy (CBT) upon remission.[8] Unlike other documented cases, our case report highlights a longer duration of psychotic symptoms following chronic inhalant use.

Gamma-aminobutyric acid (GABA) binding to glutaminergic cortical pyramidal neurons results in glutamate inhibition. The GABA disinhibition hypothesis observed in chronic inhalant use disorders may result in glutaminergic excess in midbrain projections, leading to a hyperdopaminergic state in the mesostriatum and direct pathways from the prefrontal cortex to the midbrain. This mechanism may explain the emergence of psychotic symptoms reported in our case, consistent with the hyperdopaminergic theory of schizophrenia and other

psychotic disorders.

Decreased GABA activity may also be explained at the molecular level through the parvalbumin hypothesis, wherein reduced glutamate decarboxylase 67 (GAD67) mRNA expression may be selective for the parvalbumin-containing subgroup of GABA neurons in the prefrontal cortex. Decreased parvalbumin mRNA expression is associated with the decreased density of glutamate decarboxylase 67 (GAD67) mRNA-positive GABAergic neurons. A reduction in GABAergic activity in the cortical areas could lead to disturbances related to emotional and cognitive functioning, which may underlie symptoms including social withdrawal and apathetic behavior, as observed in the above case presentation. In addition, abnormalities in the superior temporal gyrus could contribute to deficits in auditory processing and auditory hallucinations, which may explain the worsening of psychotic symptoms following acute inhalant use.[12]

Apart from the GABA hypothesis, acute inhalant use also causes NMDA receptor inhibition through a rapid, non-competitive, complete, and reversible mechanism.[13,14] Followed by increased NMDA-evoked response along with GABA dysregulation and parallel upregulation of NMDA receptors NR2A and NR2B, resulting in a sudden hyper-glutamatergic excitable state similar to the alcohol withdrawal symptoms.[15] The subsequent release of serotonin and dopamine in the caudate and nucleus accumbens in response to this excitatory state may be a leading trigger for oxidative stress, which can lead to excitotoxic-induced damage, particularly affecting myelin and white matter, following prolonged inhalant use.[13]

Solvent abuse during early adolescence leads to altered medial prefrontal cortex, accompanied by an increase in sensitivity to drug-induced excitatory synaptic alterations in the mesolimbic dopamine neurons. Thus, inhalant abuse at a younger age can predispose, perpetuate, and precipitate a psychotic illness.[9]

Planning holistic approaches at the community level to address rising trends is just as important as developing treatment guidelines. Many countries, including Australia and the United States, have implemented innovative strategies such as reformulation of products (modifying the composition of products to replace harmful chemicals with safer alternatives), deterrent additives (adding strong/foul smelling chemicals to discourage inhalation), and retailer education and restrictions training store owners to recognize and restrict sales of inhalants to minors), which have helped reduce access.

Although Australia was successful, well-developed countries such as the United States still face challenges due to inconsistent enforcement of laws at the state and federal levels and high availability of alternatives.[16] Taking valuable insights from other countries, specific combination of strategies at three different levels, the government (consistent stringent rules at state and national levels, restriction of sale to minors, and community and school-based interventions), producers (adding deterrents and replacing harmful chemicals with safer alternatives), and consumers (education about consequent harms and legal implications), can be implemented in India.

Conclusion

The paucity of Indian literature may be attributed to a lack of awareness among the general population and health professionals regarding the abuse/dependence potential and effects of inhalants.[3] The diagnosis of inhalant abuse can be difficult and relies almost entirely on clinical judgment due to a lack of exclusive withdrawal symptoms or intoxication effects with symptoms ranging from behavioral and neuro-psychiatric manifestations to mild to severe physical manifestations, the severe forms of which may lead to sudden death.[17,18] Although earlier mechanisms suggested that neuropsychiatric manifestations arise from their simple molecular structure, non-polarity, and action through nonselective disruption of lipid membrane. current studies focus on the specific targets of volatile solvents at receptor, genetic, and neurotransmitter levels, which requires further research regarding symptomatology and etiology.[1] The alternation of synaptic neuro transmissions in different brain pathways, mainly through GABA inhibition, is the core of the pathogenesis and a consistent finding in many studies. The limited existing evidence suggests that supportive treatment, such as no reversal agents or standard guidelines, currently exists; different treatment regimens have been tried with antipsychotics alone and in combination with mood stabilisers.[17,19] Overall, combining pharmacotherapy and behavioral interventions is the key. In order to decrease experimentation with inhalants, regulation without education and awareness is not enough. Future directions for upcoming researchers should focus on conducting larger surveys and more epidemiological studies. We propose including inhalants under existing drug laws and implementing tiered policies and punishments for producers, retailers, and the government to curb the rise of new drugs such as inhalants.

Authors' Contribution

Conceptualization: Sangha Mitra Godi, Vijaya Chandra Reddy Avula.
Data curation: Vundavalli Jaya Harikha.
Formal analysis: Sree Pruthvi Mithra Raman.
Investigation: Vundavalli Jaya Harikha, Sree Pruthvi Mithra Raman.
Methodology: Vundavalli Jaya Harikha.
Project administration: Sangha Mitra Godi.
Resource: Vundavalli Jaya Harikha.
Software: Vundavalli Jaya Harikha.
Supervision: Vijaya Chandra Reddy Avula, Sangha Mitra Godi.
Validation: Vijaya Chandra Reddy Avula, Sangha Mitra Godi.
Visualization: Sangha Mitra Godi.

Writing -original draft: Vundavalli Jaya Harikha.
Writing - review & editing: Vundavalli Jaya Harikha, Sangha Mitra Godi, Sree Pruthvi Mithra Raman.

Competing Interests
All authors declare that there are no competing interests or sources of funding/fees from esteemed organizations that may profit from our publication.

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Data is saved and stored with the primary author using strict password protection. Patient confidentiality is strictly observed.

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Informed Consent
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