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Mania with psychotic symptoms in an adolescent patient associated with ashwagandha use: Case report

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Abstract

Withania Somnifera (WS), known as "ashwagandha" has gained popularity as a medicinal herb used to treat various conditions including depression, anxiety, and insomnia. Biochemical studies suggest that with anolides increase the levels of GABAA, GABAB and serotonin receptors mimicking the effect of antidepressants. However, there is limited evidence regarding its effects on adolescents especially those who have yet to be diagnosed with a mood disorder.

We present a case of a 15-year-old female who developed sudden-onset manic symptoms with psychotic features, possibly precipitated by extremely high-dose Withania Somnifera (WS) use. She had ingested approximately 12,000 mg of WS over 10 days to help with mood and anxiety, and soon presented to the ED with insomnia, social withdrawal, disorganized behavior, heightened anxiety, paranoia, and racing thoughts. Prior to WS use, she was functioning well—social with peers, sleeping normally, and doing well in school. She was treated with risperidone with improvements in her psychiatric symptoms. This case highlights the importance of screening for herbal supplement use and advising caution with unregulated medicinal products, particularly in pediatric patients.

Keywords: Mania; Psychosis; Ashwagandha; Herbal supplements; Child psychiatry.

Introduction

Withania Somnifera (WS), commonly known as "ashwagandha" or "asgandh," is an evergreen shrub found in Asia, Africa, and Europe. Traditionally used as a medicinal herb in Ayurvedic medicine, its use can be traced back to 6000 BC [1]. In recent years, *ashwagandha* has gained popularity worldwide and has been used for conditions including arthritis, asthma, goiter, neurological disorders, and psychiatric disorders [1]. In 2019, it was listed as the fifth top-selling herb in the U.S., grossing over $10 million through mainstream

channels (grocery stores and drug stores) and $13 million through natural channels (e.g., supplement and specialty retail outlets) [2].

Since 2019, more than 40 human trials have investigated various uses of *ashwagandha* for short-term periods of up to approximately three months [3]. One study showed 1,000mg/day of WS improved depressive and anxious symptoms in patients with schizophrenia [4]. Another study showed significant improvement in memory, reaction time, and social cognition in patients with bipolar disorder who received 500mg/day of *ashwagandha* as an adjunctive therapy to their current medication regimen [5]. However, to date, there is insufficient evidence regarding the safety of long-term *ashwagandha* use in adult populations, and there are no documented cases concerning its effects on children/adolescents. Given the easy accessibility of *ashwagandha* among adolescents and the increasing number of youths turning to herbal supplements for natural remedies for stress, anxiety, and other health concerns, the potential for serious adverse effects is significant [6]. We present a case of sudden onset manic symptoms with psychotic features that was likely precipitated by *ashwagandha* use in a 15-year-old Hispanic female, highlighting the urgent need for educating both adolescents and their guardians about the potential risks associated with unregulated herbal supplements. Informed consent was provided by the patient's legal guardian.

Case Presentation

We present a case of a 15-year-old Hispanic female patient with a past psychiatric history of anxiety with one prior inpatient psychiatric hospitalization, who presented to the Emergency Department (ED) for psychiatric evaluation due to possible catatonia.

Approximately one month prior to presentation, the patient ingested an estimated 59 ashwagandha gummies (200 mg WS extract each) over 10 days (total dose ≈ 12,000 mg) to alleviate anxiety and improve mood. She reported using the supplement to avoid seeking medical care due to stigma surrounding mental health. She denied any recreational substance use. Recent psychosocial stressors included academic pressure, concerns about peer judgment, and a recent breakup with her boyfriend.

Within days of completing the 10-day ashwagandha course, the patient developed insomnia, social withdrawal, decreased appetite, disorganized behavior, and increasing anxiety and paranoia. During a three-day period in early August 2024, she slept less than two hours per night and exhibited religious preoccupation, paranoia ("Why are you picking me up?" from school, thoughts that people were watching her/recording her), hypervigilance toward her mother's wellbeing (concerns her mother was choking), mood lability, racing thoughts, and a brief period of pressured speech. Prior to this episode, she was described as social, high-functioning academically, and maintaining regular sleep and eating habits.

She initially presented to the ED and was diagnosed with depression with brief psychosis, then discharged home with hydroxyzine. Two days later, her symptoms worsened and she was voluntarily admitted to an inpatient psychiatric hospital for possible catatonia. Her mother noted "big eyes," a fixed arm posture, and delayed responses. The patient received an oral lorazepam challenge with minimal improvement and was started on olanzapine 10 mg nightly. On hospital day 4, her mother signed her out

against medical advice. She was discharged home on lorazepam and olanzapine for brief psychotic disorder with catatonia.

At home, the patient discontinued olanzapine for eight days due to side effects (excessive drowsiness, constipation, urinary difficulty, increased appetite). Initially, she appeared stable in a low-stimulus environment; however, she soon developed emotional lability, tearfulness, guilt, paranoia that her mother was in danger, and concerns that "doctors were going to turn me into a boy." She resumed olanzapine and followed up with an outpatient psychiatrist, who observed confusion, daily crying spells, and hypersensitivity to noise and television, with ideas of reference from TV. Her olanzapine regimen was adjusted to 5mg twice daily. Differential diagnoses included unspecified psychotic disorder, substance (ashwagandha)-induced psychotic disorder, generalized anxiety disorder, and unspecified depressive disorder.

At the time of current psychiatric consultation in the ED (late August 2024), the patient demonstrated slowed speech with a 5–6 second response latency. She made intermittent eye contact and exhibited mild upper extremity rigidity, staring, and autonomic abnormalities, yielding a Bush-Francis Catatonia Rating Scale score of 6. After receiving an oral lorazepam 1mg tablet with minimal improvement, she was administered lorazepam 1 mg intravenously, after which her speech latency, rigidity, and eye contact improved. Urine drug screen and heavy-metal panels (lead, arsenic, mercury, chromium, cadmium) were negative, and thyroid, basic metabolic, and hematologic panels were within normal limits.

Family history was notable for a maternal grandfather with schizophrenia and a mother diagnosed with bipolar I disorder with psychotic and catatonic features, who had been hospitalized three times within four months and responded to valproic acid, risperidone, and clonazepam.

Therapeutic interventions

Given the patient displayed constricted affect, delayed speech latency, slow movements, intermittent eye contact, mild upper extremity rigidity, intermittent tachycardia, and mildly disorganized speech, with improvement in symptoms following lorazepam IV challenge, her symptoms were likely related to catatonia. Therefore, the patient was discharged from the ED with oral lorazepam 1.5 mg three times per day and discontinued olanzapine. Of note, there is a temporal relationship between the patient's increased ashwagandha supplement use and initial acute onset of symptoms of social withdrawal, rapid speech, paranoia, decreased sleep, and catatonic features approximately one month ago; therefore, we postulate the excessive use of this supplement likely contributed to manifestation of these manic and/or psychotic symptoms.

Follow-up and outcomes

The patient was seen by her outpatient psychiatrist following this psychiatric consultation in the ED where she displayed improved symptoms of catatonia; however, continued to endorse paranoia, displayed thought blocking, and mood lability. She started on risperidone 0.5 mg and titrated up to 1mg to address these persistent symptoms of psychosis.

Discussion/Conclusion

The therapeutic properties of ashwagandha are attributed to a class of steroidal lactones known as withanolides [7]. Biochemical studies suggest that withanolides enhance γ-aminobutyric acid ($GABA_A$, $GABA_B$) and serotonin receptors, thereby exerting antidepressant-like effects [7]. Similar to conventional antidepressants such as selective Serotonin Reuptake Inhibitors (SSRIs), these serotonergic effects may, in susceptible individuals, precipitate mood elevation. SSRIs have been hypothesized to induce mania through receptor overstimulation, which disrupts the equilibrium between serotonergic and dopaminergic neurotransmission [8]. Rapid increases in serotonin levels can activate specific receptor subtypes, and prolonged serotinergic transmission may result from desensitization of somatodendritic 1A autoreceptors in the midbrain raphe [8]. This increase in serotonin transmission can indirectly stimulate dopaminergic activity [9]. This imbalance between serotonin and dopamine can shift mood regulation toward mania in individuals with underlying bipolar disorder. The authors postulate that this patient's strong family history of bipolar and psychotic disorders may have predisposed the patient to antidepressant-induced mania. Given that with anolides mimic antidepressant mechanisms, clinicians should be cautious of potential manic induction in individuals with bipolar predisposition who use ashwagandha or similar herbal supplements [10,11].

Although only 1–3% of minors are formally diagnosed with mood disorders, more than 50% of parents provide supplements to their children, with approximately ~37% doing so without medical guidance [12,13]. This case highlights the importance of counseling patients and caregivers about the psychiatric risks associated with herbal supplement use. Although there may have been some limitations to this case such as medication non-compliance, improvement in the patient's psychiatric symptoms were observed with risperidone monotherapy. Given the limited evidence regarding the safety and efficacy of ashwagandha in mental health, it should be used with extreme caution, as high doses may exacerbate underlying psychiatric vulnerability.

Declarations

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