

## Vertigoheel induced psychosis: A patient case report

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### Abstract

**Objective:** To describe a case of a patient who developed psychosis after ingestion of Vertigoheel for treatment of dizziness.

**Case Summary:** A 28-year-old male with no psychiatric history presented with 5 days of worsening depression and psychosis. He denied current use of prescription medications, alcohol, or illicit substances. Approximately 2 weeks prior, while visiting family in Germany, he developed dizziness. A provider in Germany prescribed Vertigoheel, 1 tablet to be taken every hour until symptom improvement. This did not improve his dizziness but did cause him to feel as if he were “in a dream.” He stopped taking the medication after 2 days but continued to feel amotivated with decreased appetite and insomnia. Several days later, he developed ego-dystonic auditory hallucinations. He returned to the United States; was admitted to an inpatient psychiatric unit for 4 days; and given olanzapine 5 mg at bedtime, lorazepam 1 mg every evening, and melatonin 6 mg every evening. He experienced gradual improvement in symptoms and was discharged with olanzapine 5 mg daily and outpatient follow-up.

**Discussion:** Vertigoheel is a homeopathic preparation containing *ambra grisea*, *Cocculus indicus*, *Conium maculatum*, and petroleum. Psychosis was not reported in any of the randomized controlled trials evaluating the use of Vertigoheel for treatment of vertigo. A literature search revealed no published reports of psychosis as a result of administration of any components of Vertigoheel.

**Conclusion:** A possible causal relationship was observed between the homeopathic supplement Vertigoheel and an acute episode of psychosis in a young male patient with no comorbidities.

**Keywords:** Vertigoheel, homeopathic medicine, psychosis

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### Background

Vertigoheel is a homeopathic preparation containing *ambra grisea* (6X), *Cocculus indicus* (4X), *Conium macula-*

*tum* (3X), and petroleum (8X).<sup>1</sup> Homeopathic dilutions are designated by either an X or a C with X dilutions being 1:10 and C dilutions 1:100. For example, 3X indicates that the active ingredient is 1 part in 1000 parts diluent.<sup>1</sup> This product is most often used to treat vertigo and motion sickness.<sup>1</sup> According to the product labeling, each tablet contains 30 mg of *ambra grisea*, 210 mg *Cocculus indicus*, 30 mg *Conium maculatum*, and 30 mg of petroleum.<sup>2</sup> Some formulations may also contain 1.5 mg of magnesium stearate, which is likely used as a lubricating agent in the manufacturing of the tablets and is classified as generally recognized as safe by the FDA.<sup>3</sup> Package directions recommend 1 tablet 3 times daily and, in acute

cases, 1 tablet every half- to 1 hour, up to 12 times per day.<sup>2</sup> Vertigoheel is available to purchase in the United States through online retailers, such as Amazon.com, Inc.<sup>4</sup>

The controversial practice of homeopathy is based on the philosophy that miniscule exposure to a noxious substance that can cause similar symptoms will stimulate self-healing in an ailing individual.<sup>5</sup> As such, the ingredients of some homeopathic preparations might raise alarm. *Ambra grisea*, or ambergris, is a substance found in the liver and intestines of the sperm whale, and although its effects are largely unknown, it claims to treat vertigo.<sup>6</sup> *Cocculus indicus*, or levant berry, is a fruit-bearing plant. The fruit is the source of picrotoxin, a chemical traditionally used as fish poison. Its use leads to stimulant effects mediated by noncompetitive GABA<sub>A</sub> receptor antagonism.<sup>6,7</sup> *Conium maculatum*, or poison hemlock, is the substance best known for killing Socrates. It can be fatal when ingested in larger than 100- to 300-mg doses, and its effects appear to be mediated by coniine, an alkaloid derivative of *C maculatum*, via nicotinic receptor antagonism.<sup>6,8,9</sup> Finally, petroleum is a semisolid mixture of hydrocarbons, and its mechanism of action is unknown.<sup>6</sup> One study<sup>10</sup> demonstrates the effect of Vertigoheel as a vasorelaxant, possibly playing a role in the treatment of vertigo. We report a case of a patient who developed acute psychosis after ingesting approximately 20 Vertigoheel tablets for treatment of new-onset vertigo.

## Case Report

A 28-year-old male with no previous psychiatric history presented to inpatient psychiatry for depressive symptoms and psychosis. Approximately 2 weeks prior to admission, he traveled to Germany to visit family when he developed new-onset dizziness and went to see a physician. He was given Vertigoheel and instructed to take 1 tablet every hour until symptoms resolve. He estimated he took about 20 tablets in 2 days.

Initially, his dizziness was reduced; however, he then started to feel like he was “in a dream.” He also started staying in bed more, feeling “no motivation to do anything.” Given these symptoms he self-discontinued Vertigoheel after 2 days. However, his lack of motivation remained. In the following days, he noticed decreased sleep and appetite. Seven days after stopping Vertigoheel and 5 days prior to admission, his dizziness returned, so he took 1 additional tablet. He then began to hear a voice stating, “You’re sick, you’re crazy, you’re not going to get better.” The voice began to command him to “hit people” and “to break things.” It occasionally told him to “kill yourself.” He could not identify the voice and felt it was “inside his head” rather than in the room with him. The

commands were ego-dystonic as he had no intention of harming anyone or himself.

He asked his father to help arrange a flight back to the United States and immediately presented to psychiatric services upon return. On admission, he endorsed insomnia (about 1 hour of sleep each night), poor appetite, amotivation, hopelessness, fatigue, and poor concentration that he had been experiencing for 1.5 weeks. He denied actually feeling sad or suicidal and denied history of depressive symptoms. He stated the auditory hallucinations were constant. He also felt anxious, noting symptoms of racing thoughts, restlessness, and muscle tension. His urine drug screen was negative, and admission labs were unremarkable. He denied current use of prescription medications, alcohol, or illicit substances other than marijuana on a single occasion 1 month prior to admission. He did endorse smoking tobacco cigarettes, half a pack per day for years. He denied any other physical symptoms on a 10/14 review of systems. He was employed as a cab driver with a high school diploma.

On admission, he was started on olanzapine 5 mg every night and lorazepam 1 mg every night for sleep. Olanzapine was chosen for its sedating and appetite-stimulating effects. He found the lorazepam to be too sedating; thus, it was discontinued in the hospital. He was able to get 6 to 7 hours of sleep nightly, and auditory hallucinations gradually resolved. However, he was still having depressive symptoms. He was discharged after a total of 4 days with a prescription for olanzapine 5 mg at bedtime and follow-up in the crisis support clinic in 2 days.

At his follow-up visit, he reported still feeling depressed and was prescribed citalopram 10 mg daily. One month later, he endorsed drowsiness from his first and only dose of citalopram and was switched to sertraline 25 mg daily. Around the same time, olanzapine was tapered to 2.5 mg every night as he was feeling “groggy” in the mornings. He was seen shortly after in the emergency department with symptoms of a panic attack. His labs, vitals, and electrocardiogram were normal, and sertraline was discontinued. Soon after, he was started on buspirone 10 mg twice daily and 1 mg lorazepam daily in addition to olanzapine. One month later, buspirone was stopped, and nortriptyline 10 mg at bedtime was initiated while olanzapine and lorazepam were continued. He continued to have panic attacks. He endorsed hearing a voice before panic attacks that told him he was “going to die.” It remains unclear whether he was having auditory hallucinations during these episodes or whether he was experiencing ongoing anxiety about having another psychotic episode and interpreting an internal monologue as an auditory hallucination. He denied voices outside of panic attacks.

## Discussion

Vertigoheel has been studied in a small number of published clinical trials for the treatment of vertigo. The first,<sup>11</sup> published in 1993, did not have a comparator group. Thirty-one patients were administered 1 to 3 tablets of Vertigoheel 3 times daily for vertigo of various etiology for 6 weeks. No side effects were reported. A second study<sup>12</sup> compared Vertigoheel and betahistine for treatment of vertigo. Patients were administered 15 drops (equivalent to 1.5 tablets) of Vertigoheel 3 times daily or 18 mg/d of betahistine for 42 days.<sup>2,12</sup> No psychiatric adverse events were reported. The milligram dose of Vertigoheel per day was not reported. The final study<sup>13</sup> compared Vertigoheel with *Ginkgo biloba* for 6 weeks for treatment of atherosclerosis-related vertigo. Subjects received Vertigoheel 2 tablets 3 times daily or *G biloba* 40 mg 3 times daily. Again, no psychiatric adverse effects were observed in either group. There have been no published case reports of psychosis or hallucinations resulting from any of the individual components of Vertigoheel.

This is the first case report, to the authors' knowledge, that describes an apparent Vertigoheel-induced psychosis. A score of 4 on the Naranjo scale indicates this was a possible adverse drug reaction to Vertigoheel (2 points for adverse event appearing after the drug was given, 1 point for improvement when the drug was discontinued, 2 points for reappearance of effects when drug was readministered, -1 point for possibility of alternative causes for reaction).<sup>14</sup> As there are no previously published reports, the mechanism behind the reaction is unclear, and the following discussion is largely theoretical.

The alkaloid derivative of *C maculatum*, coniine, is structurally similar to nicotine and binds as an antagonist to nicotinic receptors.<sup>9</sup> Acute coniine toxicity leads to neuromuscular blockade via stimulation of acetylcholine receptors followed by depolarization and paralysis.<sup>9</sup> Although there are few data on the effects of coniine in the human brain, rat studies indicate that it also increases dopamine release in the striatum.<sup>15</sup> If the effects in the human brain are similar, one may speculate that increased dopamine could lead to symptoms of psychosis.

*Cocculus indicus* is a noncompetitive antagonist at GABA<sub>A</sub> receptors.<sup>7</sup> Theoretically, it may be possible for excessive GABA<sub>A</sub> antagonism to lead to an imbalance of other neurotransmitters (eg, glutamate) and ultimately induce adverse effects similar to those caused by alcohol withdrawal, including hallucinations. However, this explanation seems unlikely given the lack of other, more common, withdrawal-like symptoms (tremor, autonomic instability, etc) in this case.

Another important aspect to consider is whether the patient was receiving the ingredients included in the labeling. The German Medicinal Products Act requires that homeopathic preparations be registered as such, and the German Homeopathic Pharmacopoeia provides standards to ensure quality of manufactured products.<sup>16,17</sup> As such, it would seem appropriate to conclude that the patient likely received the ingredients described.

## Conclusion

A possibly causal relationship was observed between the homeopathic supplement Vertigoheel and an acute episode of psychosis in a young male patient with no comorbidities.

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