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Case Study

Case Report on Acute Hexaconazole Poisoning in A Chronic Alcoholic Patient

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ABSTRACT

Hexaconazole is a broad-spectrum systemic triazole fungicide widely used in agriculture to control fungal infections in crops. Although it is considered to have low mammalian toxicity, human poisoning cases remain rare and largely underreported in medical literature. The exact mechanism of toxicity in humans is not well understood, but animal studies suggest that exposure may lead to gastrointestinal distress, neurological dysfunction, and, in severe cases, respiratory and cardiovascular complications. Given the limited clinical data available on Hexaconazole poisoning, proper management guidelines are not well established, making early recognition and prompt supportive care crucial. This case report aims to contribute to the growing understanding of Hexaconazole toxicity by detailing the clinical presentation, management, and outcome of an acute ingestion case in a chronic alcoholic patient.

INTRODUCTION

Hexaconazole is a systemic triazole fungicide extensively used in agriculture to prevent and treat fungal infections in crops. It functions by inhibiting ergosterol biosynthesis, an essential component of fungal cell membranes, thereby disrupting fungal growth and reproduction⁽¹⁾. Due to its selective toxicity towards fungi, Hexaconazole is considered to have low toxicity in

mammals. However, the safety profile in humans remains largely understudied, as most toxicological data are derived from animal studies rather than human cases. Despite its widespread use in agriculture, cases of human Hexaconazole poisoning are rare, with limited clinical documentation available⁽²⁾. Unlike organophosphate and carbamate pesticides, which have well-characterized toxicological profiles and

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specific antidotes, Hexaconazole lacks a well-defined toxicological framework, making management challenging. Understanding its mechanism of toxicity, clinical effects, and appropriate treatment protocols is critical for improving patient outcomes in cases of accidental or intentional ingestion.

MECHANISM OF TOXICITY

The primary mode of action of Hexaconazole is inhibiting the enzyme lanosterol 14 α -demethylase, which is crucial in the synthesis of ergosterol, an essential structural component of fungal cell membranes⁽³⁾. This disruption leads to fungal cell membrane instability, ultimately causing fungal growth inhibition and death. However, triazole fungicides like Hexaconazole may exert unintended toxic effects in non-target organisms, including mammals. Hexaconazole has been shown to have hepatotoxic, neurotoxic, and cardiotoxic effects in animal studies, raising concerns about potential human toxicity⁽⁴⁾. Animal research indicates that Hexaconazole accumulates in fatty tissues, potentially leading to chronic exposure risks in individuals handling or ingesting the fungicide.

Clinical Manifestations of Toxicity

The toxicity of Hexaconazole in humans is not well characterized, as there is a scarcity of documented poisoning cases. Most available data are extrapolated from animal studies and acute pesticide exposure reports⁽⁵⁾. However, based on experimental evidence and clinical observations, ingestion of Hexaconazole may result in a wide range of symptoms, including:

1. Gastrointestinal Effects

- Nausea
- Vomiting
- Excessive salivation
- Abdominal pain
- Diarrhea

Gastrointestinal irritation is one of the most commonly reported symptoms following

fungicide ingestion⁽⁶⁾. The severity of these effects depends on the dose ingested and the individual's health status.

2. Central Nervous System (CNS) Effects

- Dizziness
- Drowsiness
- Altered mental status (e.g., confusion, disorientation)
- Headache
- Seizures (in severe cases)

Some studies suggest that Hexaconazole may act as a CNS depressant, leading to drowsiness, confusion, and respiratory depression in severe cases⁽⁷⁾. Neurotoxicity could be attributed to its lipophilic nature, allowing it to cross the blood-brain barrier.

3. Hepatic Effects (Liver Toxicity)

- Elevated liver enzymes (AST, ALT)
- Hepatocellular injury
- Jaundice (in severe cases)

Liver damage has been observed in rats and mice exposed to Hexaconazole⁽⁸⁾. The exact mechanism of hepatotoxicity in humans remains unclear, but evidence suggests oxidative stress and mitochondrial dysfunction may contribute to liver cell damage.

4. Cardiovascular Effects

- Hypotension (low blood pressure)
- Tachycardia (rapid heartbeat)
- Cardiac arrhythmias (irregular heartbeats)

In animal studies, Hexaconazole exposure was linked to alterations in cardiac function, possibly due to direct myocardial toxicity or secondary effects of systemic toxicity⁽⁹⁾.

Comparison with Other Pesticides

Unlike organophosphate and carbamate pesticides, which have well-documented toxicological profiles, Hexaconazole poisoning is less understood. Organophosphate poisoning, for example, results in cholinergic toxicity with symptoms such as salivation, muscle twitching, and respiratory distress. In contrast, Hexaconazole

toxicity lacks a clear mechanism of poisoning in humans, making its clinical presentation more unpredictable. Additionally, organophosphate poisoning has a specific antidote (atropine and pralidoxime), while no antidote exists for Hexaconazole poisoning. This makes supportive care the primary treatment strategy⁽¹⁰⁾.

CASE PRESENTATION

Mr. M, a 39-year-old male patient, was admitted to the male medical ward on November 2, 2024, following deliberate ingestion of approximately 50 mL of unknown poison at home around 1 PM, with c/o breathlessness, salivation, and four episodes of vomiting. On examination, he was conscious, oriented, afebrile, and his vitals were B.P. 110/40 mmHg, PR 86/min, SpO₂ 98%, RR 16/min, CVS S1S2 (+), CNS NFND, RS B/L AE+, and P/A soft BS+. Later they confirmed that he had consumed Hexaconazole 5% fungicide. He had no medical, medication, or surgical histories, with no known allergies or contributory family, but he is a chronic alcoholic person. In the ER, they performed stomach wash, going with nil per oral (NPO), and started with 2 pints of NS, RL, and 1 pint of DNS at 75 ml per hour, Inj. Pantoprazole 40 mg IV BD, Inj. Emeset (Ondansetron) 4 mg IV BD, and Inj. Atropine 0.6 mg IV SOS, with normal lab reports. The same treatment was continued. On Day 2, that is, 3rd Nov, 2024, and then the patient was referred to a psychiatrist. and then Inj. Thiamine 200 mg in 100 ml NS IV OD, Inj. Vit. B12 IM OD, and Inj. Lorazepam 4 mg IM SOS were added to the treatment. On Day 3, that is, on 4th Nov, his vitals were normal, and the IV fluids were discontinued. On Day 4, that is, on 5th Nov, Inj. Pantoprazole and Inj. Emeset (ondansetron) were changed to tablet form the same day the patient was discharged. on DOR (Discharge on Request).

DISCUSSION

Hexaconazole is a triazole fungicide widely used in agriculture due to its potent antifungal activity, primarily achieved by inhibiting ergosterol

biosynthesis, a key component of fungal cell membranes. While it is considered to have low mammalian toxicity, its effects on human health, especially in cases of acute poisoning, remain poorly documented. The limited available data suggest that Hexaconazole poisoning does not have a well-defined clinical syndrome, and its toxic effects may be variable, depending on the dose ingested and the individual's physiological condition. Toxicity Profile and Potential Mechanisms of Action Experimental studies on animals have demonstrated that Hexaconazole exposure can lead to systemic toxicity, particularly affecting the gastrointestinal, nervous, and respiratory systems. Reported symptoms following ingestion include:

- Gastrointestinal distress: Nausea, vomiting, excessive salivation, and abdominal discomfort due to mucosal irritation.
- Respiratory involvement: Breathlessness and pulmonary irritation, potentially due to chemical pneumonitis if aspiration occurs.
- Neurological effects: Central nervous system (CNS) depression, confusion, dizziness, and in severe cases, altered sensorium or seizures.

While triazole fungicides do not share the same toxicity mechanism as organophosphates or carbamates, some reports suggest that Hexaconazole may induce mild cholinergic-like symptoms due to its potential effect on acetylcholine metabolism. However, this remains speculative and requires further research.

Currently, there is no specific antidote for Hexaconazole poisoning, making management entirely supportive and symptomatic. In this case, the patient presented with breathlessness, excessive salivation, and vomiting following ingestion of approximately 50 mL of Hexaconazole 5% fungicide. Immediate gastric decontamination with a stomach wash was performed to reduce systemic absorption. The patient was kept nil per oral (NPO) to prevent



further gastric irritation, and intravenous (IV) fluid support was initiated to maintain hydration and electrolyte balance. Proton pump inhibitors (PPIs) and antiemetics were administered to protect the gastric mucosa and control nausea and vomiting, respectively. Atropine was available for use on a symptomatic basis (SOS), as it may be beneficial in managing excessive salivation or suspected cholinergic symptoms.

Role of Alcohol Dependence in Poisoning Cases

A key aspect of this case is the patient's history of chronic alcohol use, which may have influenced both the risk of self-poisoning and his physiological response to toxic ingestion. Chronic alcoholism is associated with:

- Increased risk of deliberate self-harm: Alcohol dependence is strongly linked to psychiatric disorders, impulsivity, and suicidal behaviour, making psychiatric evaluation a crucial component of care.
- Nutritional deficiencies: Long-term alcohol consumption leads to thiamine (vitamin B1) and vitamin B12 deficiencies, increasing the risk of Wernicke's encephalopathy and other neurological complications.
- Altered drug metabolism: Chronic alcohol use can either enhance or reduce the metabolism of xenobiotics, potentially modifying the toxicokinetics of ingested substances.

CONCLUSION

This case highlights the importance of early recognition and prompt supportive care in managing acute Hexaconazole poisoning. Although Hexaconazole is generally considered to have low mammalian toxicity, ingestion can lead to significant gastrointestinal distress, respiratory symptoms, and potential neurological effects, necessitating urgent medical intervention. Given the absence of a specific antidote, treatment remains supportive, with emphasis on gastric decontamination, fluid resuscitation, symptom control, and close monitoring of vital parameters.

Furthermore, the patient's history of chronic alcohol use played a crucial role in both the risk of self-poisoning and subsequent management considerations. The addition of thiamine, vitamin B12, and psychiatric evaluation was essential in addressing underlying nutritional deficiencies and mental health concerns. This underscores the need for a multidisciplinary approach, incorporating toxicology, internal medicine, and psychiatry, particularly in cases involving intentional self-harm and substance dependence. The patient showed progressive clinical improvement with appropriate medical management and was discharged in a stable condition upon request. However, this case emphasizes the necessity of long-term psychiatric follow-up and alcohol rehabilitation to prevent recurrent self-harm episodes. Given the limited data on human Hexaconazole toxicity, further research and case studies are needed to develop comprehensive treatment protocols and improve clinical outcomes in future cases.

CONFLICTS OF INTEREST

The authors declare no conflict of interests.

AUTHORS CONTRIBUTION

All authors have contributed equally to this work and have read and approved the final version of the manuscript.

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