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Case Report

A case study on seizure induced by antkiller poisoning

Karthikeyan K, Ms. Girija S, Ms. Keerthana GC, Jagadeesan M

SRM College of Pharmacy SRM Institute of Science and Technology

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Corresponding author: Jagadeesan M, Assistant Professor, SRM College of Pharmacy SRM Institute of Science and Technology. Email: jagapharmd2013@gmail.com

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Abstract

Pesticide poisoning, which occurs due to occupational, accidental, and intentional exposure, is a serious public health issue. However, it has been reported that deliberate self-poisoning by pesticides causes a higher mortality rate. Gammaxene or gamma benzene hexachloride (BHC) is commonly used as an ant killer. On oral ingestion in humans, it causes vomiting, agitation, confusion, and seizures. Here we report the case of a 25-year-old male patient admitted to the hospital with complaints of vomiting and throat pain after attempting suicide by consuming gammaxene. After 32 hours, the patient developed seizures. A magnetic resonance imaging (MRI) investigation showed superior sagittal vein thrombosis. He was treated with phenytoin. His condition stabilized immediately after the administration of phenytoin. He was discharged after three days and put on maintenance therapy of levetiracetam. The patient was healthy and stable on follow-up after a week.

Keywords: ant-killer; gammaxene; seizure; sagittal vein thrombosis

Introduction

Intentional self-poisoning with pesticides in the Asia-Pacific region is estimated to result in 300,000 deaths annually.[1-5] The effect and mode of action of organochlorine insecticides were elucidated in the early 1980s. The impact of the toxicity of organochlorine and organophosphorus pesticides and insect resistance has played a significant role in recent years in developing other families of insecticides with less toxicity.[6,7] Cyclodienes (e.g., endosulfan) and cycloalkanes (e.g., gammaxene) are the two main classes of insecticides that inhibit $GABA_A$-gated chloride channels, thereby producing neuronal hyperexcitability. Dermatitis, seizures, ataxia, and confusion are the common manifestations of gammaxene toxicity in humans.[8] Despite its absorption, the primary toxic indication of gammaxene is the stimulation of the central nervous system (CNS). It can produce signs of CNS hyperexcitability at high doses in animals. This characteristic property has been used experimentally to induce convulsions similar to those caused by electroshock and pentylenetetrazol methods.[9] This paper discusses a case of self-poisoning with ingestion of ant killer (gammaxene) that induced seizure in a young patient.

Case report

A 25-year-old male patient working as a driver was admitted to the emergency department of the SRM Hospital at forenight 30 minutes after the ingestion of about 50 g of ant killer gamma benzene hexachloride (BHC) mixed in approximately 100 mL of water in an attempted suicide due to emotional stress. The patient complained of nausea, throat pain, and dizziness at the time of admission. The vitals were stable. The patient was subjected to a gastric lavage procedure using Ryle's tube intubation along with 50 g of activated charcoal. Intravenous fluids such as normal saline and ringer lactate (100 mL/hour) were given after gastric lavage. The patient was healthy, and his vitals remained stable. The patient's clinical history showed an on and off headache for the past one month, for which the patient took over-the-counter medicines for relief from pain.

The blood pressure and pulse rate shot up to 170/110 mmHg and 110 bpm on the second day, respectively. Metoprolol extended-release tablet

12.5 mg once a day for five days was given to the patient to manage the blood pressure. On the third day, at 5:40 a.m., the patient reported one episode of generalized tonic-clonic seizure, which lasted for 5–7 min followed by loss of consciousness for 3–5 min with rolling eyelids and tongue bite. The patient was shifted to the intensive care unit (ICU). Phenytoin 100 mg injection was administered through IV immediately at eight-hour intervals, followed by the maintenance dose of phenytoin 200 mg tablet at bedtime from the next day.

Table 1: Laboratory Investigations

Haematology	**Day 1**	**Day 3**	**Day 5**
Hb (11-16.5 g/dl)	8.6	8.3	9
PCV (35-50%)	29	28	31
Neutrophils (40-70%)		93	
Lymphocytes (15-30%)	18	9	8
Monocytes (4-8%)	3	3	1
Eosinophils (1-6%)	3	3	2
WBC (4,500 to 11,000 cells/microliter)	15800	16000	
Platelets (1-4lakh cells/cumm)	4 lakhs	2 lakhs	227000
MCV (79-93.3 fl)	63	66	
MCH (26.7-31.9 pg/cell)	20	20	
MCHC (32.3-35.9)	31	21	
Na/K (135-145 mEq/L) (3.5-5.5mEq/L)	137/3.2	136/3.4	137/3.3
Serum creatinine (0.6-1.2mg/dL)	1.1	0.9	1.0
Total proteins (6.8-8.3g/dL)		7.8	
SGOT/SGPT (5-35) (5-40) U/L		26/16	
Bilirubin total (0.5-1.1,g/dL)		0.6	
Direct bilirubin (<0.25mg/dL)		0.2	

On the fourth day, the patient was shifted to the general ward. The opinion of a neurologist was sought. A magnetic resonance imaging (MRI) investigation showed superior sagittal vein thrombosis. The patient was given Enoxaparin sodium injection 0.6 mL subcutaneous for two days, followed by acenocoumarol tablet 2mg at night for four days. Paracetamol was given whenever necessary.

The findings of laboratory investigations are shown in Table 1. Prothrombin time was found to be 15.3 seconds (control 13.5 seconds). Hematologic investigations showed decreased hemoglobin and elevated WBC count. Peripheral smear revealed moderate microcytic hypochromic anemia. On investigation, the social history of the patient revealed alcohol consumption and malnutrition. Thiamine deficiency is common in alcoholism, and inflammation of the gut reduces the ability to absorb vitamins. Hence, a thiamin 100 mg/ml (thiamine nitrate) injection was administered once daily intravenously from day 1 to day7for tackling nutritional deficiency based on subjective evidence.

On the fifth day, the patient was treated with piperacillin/tazobactam 4.5 g IV injection three times daily as a prophylaxis antibiotic due to an increase in the neutrophil and WBC count. On the sixth day, levetiracetam 250 mg tablet twice daily was given. On the seventh day, the patient was discharged with levetiracetam 250 mg twice daily and acenocoumarol 2 mg tablet at night for one week. He was asked to come for a follow-up review after one week. The patient was found to be healthy and stable at review after a week.

Discussion

Benzene hexachloride is one of the chlorinated hydrocarbon pesticide groups. It is the most potent pesticide among its isomers. Gammaxene is absorbed from gastrointestinal, respiratory, and cutaneous routes. It is poorly absorbed after a single oral dose as it induces repeated vomiting immediately after ingestion.[10] Feldman and Maibach's toxicokinetic analysis of lindane overdose revealed that the half-life of gammaxene was 26 hours in urinary excretion after intravenous dosing in healthy volunteers[11], followed by non-specific signs of nervous system stimulations such as hyperexcitability or irritability.[12] Gammaxene levels in blood plasma will usually be less than the lipid-rich tissues in the body due to gammaxene's lipophilic nature.[8] Humans require a small amount of gammaxene to induce seizures; a single oral dose of 45 mg of gammaxene is enough to trigger a seizure in

adults.[13] Tonic-clonic convulsions occur within 1–2 hours and can last for days.[14]

In the case reported here, the patient was admitted 30 minutes after the ingestion of gammaxene. He was immediately subjected to a gastric lavage procedure along with 50 g of activated charcoal. Still, the decontamination procedure using gastric lavage proved to be ineffective and had a less beneficial effect.[15] The patient developed one episode of generalized tonic-clonic seizure approximately after 32 hours of ingestion of gammaxene, which lasted for 5–7 minutes, followed by loss of consciousness and3–5 minutes of rolling up eyelids and tongue bite. The effect of seizure on the third day may be the delayed effect of gammaxene. At the time of hospital admission, gammaxene could have reached the lipid-rich tissues in the body due to its lipophilic nature.[16]

Gammaxene can cause convulsions by interfering with ammonia metabolism in the brain due to its high concentration in the white matter of the brain.[16] The mechanism of organochlorine neurotoxicity of DDTs usually involves the modulation in the opening of sodium channels, whereas other classes of substances like gammaxene act by blocking the GABA-gated chloride channel, resulting in abnormal intracellular signaling systems.[17] Dermatitis, seizures, ataxia, and confusion are the characteristic manifestations of the toxicity of gammaxene. Rhabdomyolysis, pancreatitis, or disseminated intravascular coagulation can occur very rarely.[18]

The treatment for gammaxene poisoning is not clearly defined. Symptomatic management of seizures and myocardial depression is required. Hypotension can be treated with dopamine. In some cases, oral ingestion of cholestyramine has been used to prevent the absorption of the toxin.[19] The convulsing patient who ingested gammaxene can be managed with an intravenous administration of phenobarbital (5 mg/kg of body weight) or diazepam (0.1–0.3 mg/kg).[20] A syrup of ipecacuanha can be given to produce emesis, or gastric lavage has to be done in all the suspected cases of gammaxene poisoning, followed by saline cathartics.[21]

Benzodiazepines are widely used in the treatment of stress and anxiety disorders. Recent studies by several workers show that diazepam, a representative of the benzodiazepine family, has a probable protective effect in poisoning cases with rival organophosphorus chemical agents. They have demonstrated that benzodiazepines had a protective effect in peripheral and central action, especially against convulsions caused by the central nervous system (CNS) when administered before exposure to the organophosphorus agents.[22]

The ant killer poison containing the chemical compound gamma benzene hexachloride is known to cause a seizure. In this case, the patient was diagnosed with superior sagittal vein thrombosis, which was revealed by an MRI investigation after the seizure episode. Superior sagittal vein thrombosis is a neurologic problem that can cause seizures in 40% of patients.[23] On assessing the causality using the WHO causality assessment scale, with the reasonable time relationship, it can be said that the ant poison induced seizure is a possible event. But, likely, it could also be explained by thrombosis.

Conclusion

This case study concludes that the ant-killer containing gammaxene is known to cause vomiting, agitation, and seizure in humans. The uniqueness of this case is that seizure might be induced as a delayed side effect (Type D). Hence, supportive care and specific and immediate management are needed to avoid complications of seizures caused by ant killer poisoning. Benzodiazepines can be given to prevent the early onset of seizures. Additional care to the patients can prevent the severity of toxicity and improves the patient's quality of life.

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References

1. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. Wld Hlth Statist Quart. 1990;43:139–44.
2. Van der Hoek W, Konradsen F, Athukorala K, Wanigadewa T. Pesticide poisoning: a major health problem in Sri Lanka. Soc Sci Med. 1998;46:495–504.
3. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. Q J Med. 2000;93:715–31.
4. Gunnell D, Eddleston M. Suicide by intentional ingestion of pesticides: a continuing tragedy in

developing countries. Int J Epidemiol. 2003; 32:902–9.
5. Eddleston M, Phillips MR. Self-poisoning with pesticides. BMJ. 2004;328:42–4.
6. Bloomquist JR. Toxicology, mode of action, and target site mediated resistance to insecticides acting on chloride channels. Comp Biochem Physiol. 1993;106C:301–14.
7. Bloomquist JR. Ion channels as targets for insecticides. Annu Rev Entomol. 1996;41:163–90.
8. Nolan K, Kamrath J, Levitt J. Lindane toxicity: a comprehensive review of the medical literature. Pediatr Dermatol 2012;29(2):141–6.
9. Litterest CL, Miller E. Distribution of lindane in brains of control and phenobarbital pretreated dogs at the onset of lindane induced convulsions. Bull Environ Contam Toxicol 1975;13:619-624.
10. Starr HG Jr. Clifford NJ. Acute lindane intoxication: A case study. Arch Environ Health 1972;25:374-375.
11. Wiles DA, Russell JL, Olson KR, Walson PD, Kelley M. Massive lindane overdose with toxicokinetics analysis. J Med Toxicol. 2015; 11(1):106-9.
12. Falk W, Hinrichs R. Jacutin poisoning in childhood. Med Klin Wschr 1957;42:1837.
13. Hayes WJ. Clinical Handbook of Economic Poisons. Atlanta, US Communicable Disease Center. Washington, DC: U.S. Government Printing Office, Public Health Service Publication No. 476.1963.p.52-55
14. Eskenasy JJ. Status epilepticus by dichloro diphenyl trichloroethane and hexachloro cyclohexane poisoning. Roum Rev Neurol 1972;9:436-452.
15. Vale JA, Kulig K, American Academy of Clinical Toxicology., European Association of Poisons Centres and Clinical Toxicologists. J Toxicol Clin Toxicol. 2004;42(7):933-43.
16. St Omer VV. Investigations into mechanisms responsible for seizures induced by chlorinated hydrocarbon insecticides –The role of brain ammonia and glutamine in convulsions with the rat and cockerel. J Eurochem 1971;8:356-74.
17. Kanavouras K, Tzatzarakis MN, Mastorodemos V, Plaitakis A, Tsatsakis AM. A case report of motor neuron disease in a patient showing a significant level of DDTs, HCHs, and organophosphate metabolites in hair as well levels of hexane and toluene in blood. Toxicology and Applied Pharmacology 2011;256(3),399-404.
18. Valjak B, Fabecic-Sabadi V, Oberiter V. Gammacid poisoning in a child (Otrovanjegamacidomkoddjetata). Lijecn Vjesn 1966;88:157-160.
19. Goel A, Aggarwal P. Pesticide poisoning. Natl Med J India 2007;2013:182–91.
20. Schmid A. Proceedings: Investigations into the suitability of oxazepam and diazepam as antidotes against gammahexachlorocyclohexane (lindane). Arch Pharmacol 287 suppl R100:1975.
21. Toxic effects of technical benzene hexachloride and its principal isomers. J Am Med Assoc. 1951;147(6):571-4.
22. Tsatsakis AM, Manousakis A, Anastasaki M, Tzatzarakis M, Katsanoulas K, Delaki C, Agouridakis P. Clinical and toxicological data in fenthion and omethoate acute poisoning. Journal of Environmental Science and Health, Part B, 1998;33(6),657-670
23. Gupta RK, Jamjoom AA, Devkota UP. Superior sagittal sinus thrombosis presenting as a continuous headache: a case report and review of the literature. Cases J. 2009;2:9361.