ejpmr, 2023, 10(10), 365-366

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Study

ISSN 2394-3211

EJPMR

CASE REPORT ON RODENTICIDE POISONING

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Article Received on 12/08/2023 **Article Revised on 02/09/2023** **Article Accepted on 23/09/2023**

ABSTRACT

Rodenticides or "rat poisons" are mixed compounds used to eradicate rodents. Rodenticide toxicity is one of the common modes of poisoning encountered in our part of the country. It is cheap and easily available in the market, in various forms such as powder, cake, paste etc. Rodenticide toxicity can range from asymptomatic presentation to life threatening complications and death, based on the amount and type of rodenticide ingested. Here is the case report of a 47 year old male who had suicidal consumption of rodenticide.

KEYWORDS: Rodenticides, Bromadiolone.

INTRODUCTION

Rodenticides or "rat poisons," are chemical agents used to eliminate rodents including rats, mice, beavers, squirrels etc. Since India is predominantly an agricultural nation, rodenticides are both widely utilized and easily accessible. They are available in various formulations as powders, pastes, pellets, cereal baits, or blocks. An ideal rodenticide is one that is highly poisonous to rodents in small doses, non toxic to animals that are not its target, and resistant to bait shyness and bait refusal.[1]

Rodenticides are classified into three groups: first generation anticoagulants, second generation anticoagulants (SGARs), and acute toxicants. First generation anticoagulant rodenticides last for approximately 7 days in an animal's system, but SGARs are more potent and generally last for 4 weeks. The third category, acute toxicants, include phosphorus, thallium, zinc phosphide etc.[2]

Bromadiolone is a second generation 4-hydroxycoumarins commonly referred to as super warfarin (Newer anticoagulant group of rodenticides called "superwarfarins").[3]

The mechanism of intoxication with coumarin anticoagulants is via a specific inhibition of blood coagulation. Vitamin K is needed for the functional synthesis of coagulation factors II, VII, IX and X. Blood vessels lose their elasticity, and subsequently rupture of large blood vessels occur, clinically manifested by massive hemorrhage and hematoma.[4]

In India, however, the common agents implicated are the metal phosphides, particularly aluminum phosphide. There has been 12,886 cases of exposure to rodenticides documented in the National Poison Data System of the American Association of Poison Control Centers. It was also noted that anticoagulants contributed to around 82%–89% of all rodenticide poisoning. Out of these anticoagulants, second-generation anticoagulants, such as bromadiolone, contributed 83%–91% of all anticoagulant poisonings.[5]

The diagnosis of rodenticide intoxication has to be considered for any patient with increased INR, prolonged activated partial prothrombin time. The intoxication can be confirmed by the identification and measurement of the rodenticides in plasma by specific assays. Immediate administration of high doses of phytomenadione (vitamin K1) and/or factor prothrombin complex concentrate (30UI/kg) can successfully reverse the anticoagulant effects of anticoagulant rodenticides. With tissue half-lives estimated at 16 days, reversal of superwarfarin toxicity is a long-term issue. Therefore, long-term daily treatment for several weeks of phytomenadione is necessary. To avoid re-bleeding, close monitoring of INR is necessary.[6]

We herein discuss a case of a 47 year old man who took rodenticide for deliberate self harm, presenting initially with mild symptoms and was managed effectively.

CASE REPORT

A 47-year-old male was brought to the Emergency Medicine Department with alleged history of suicidal consumption of rodenticide bait. He consumed the toxin by 10 am (not witnessed) and was presented to the hospital by 6pm. He had nausea but did not vomit. On arriving, RT was inserted and aspiration done showed blackish colored aspirate in small volume (~10ml). A rodenticide cover box brought by the relatives which revealed BROMADIOLONE was the chief constituent, and possible source of poisoning.

On general examination, the patient was found to be conscious and oriented to time, place and person along with heart rate was - 100/min, respiratory rate of 16 breaths/ min, blood pressure of 150/90 mm of Hg and 98% oxygen saturation on room air. Abdomen examination revealed non tender with no other significant findings. Nervous system examination did not reveal any specific focal neurological deficit. Cardiovascular and respiratory examination revealed no abnormality. His blood investigations at the time of admission revealed hemoglobin: 14.6 g/dl, total count 8600/microL, platelets were 2.43 lakhs, PT 13.5, INR 0.92, Bilirubin T/D/I 0.78/0.16/0.62, SGPT/SGOT 37/29. He was treated with N-acetyl cysteine intravenous infusion with standard loading dose of 150 mg/kg over a time period of 15 min, following which a dose of 50 mg/kg over 4 hours and then 100 mg/ kg over 16 hours. Patient was also started on dextrose fluids, multivitamins and prophylactic antibiotics.

The patient had complaints of breathing difficulty, and was started on with 3L oxygen.

The next day the patient went into bradycardia and unresponsiveness. CPR was initiated, the patient was intubated and shifted to ICU. On the second day INR was increased (1.30/1.38), Hb 13, LFT was increased. He was then given a STAT dose of Vitamin K 10mg and 2 pint FFP (NAC infusion ongoing). Patient was monitored daily for coagulopathy, liver function test and kidney function test. There was a remarkable decrease in deranged levels of AST and ALT. On day 7 the patient was hemodynamically stable and was discharged.

DISCUSSION

Bromadiolone is a 4-hydroxycoumarin derivative that shows high potency and long-acting anticoagulation by inhibiting vitamin K epoxide reductase[7] The most common clinical manifestations of bromadiolone ingestion include bleeding in various tissues and organs, with the primary cause of death being intracranial hemorrhage . Laboratory examinations commonly show noticeably prolonged APPTs and PTs and increasing INRs in bromadiolone-poisoned patients. It is noteworthy that the primary clinical manifestations may emerge several days after bromadiolone ingestion due to its long half-life, and the exact manifestations can vary extensively.[8] Vitamin K1 is an effective antidote to bromadiolone poisoning. The patient needed long-term treatment of vitamin K1 because of the long half-life period of bromadiolone.

CONCLUSION

Bromadiolone poisoning should be diagnosed and treated as early as possible. Misdiagnoses can easily occur because some patients cannot identify their contact history with bromadiolone, or clinical symptoms are atypical. Though bromadiolone is considered as safe for human beings, fatalities do occur. Based on our case report, we can conclude that bromadiolone poisoning can cause minor signs and symptoms in patients. These signs and symptoms are typically treatable with supportive care and can be managed with good early therapy.

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