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Amitraz Poisoning: A Rare Case of an Unusual Pesticide Poisoning

Shallu Chaudhary[1], Major Amit Atwal[2], Neha Atwal[3]

[1]Medical Officer, Health & Family Welfare Department, Himachal Pradesh, India
[2]Medical Officer, Military Hospital Jutogh Cantt, Shimla, Himachal Pradesh, India
[3]Junior Resident, Department of Anaesthesiology, IGMC Shimla, Himachal Pradesh, India

Corresponding Author: Major Amit Atwal

ABSTRACT

Amitraz is a pesticide used in animals and agriculture. It consists of triazapentadiene which is a centrally acting alpha2 adrenergic agonist. Amitraz poisoning is uncommon in human beings. It occurs via the oral, dermal or inhalational route. The clinical manifestations are mainly central nervous system depression, respiratory depression and cardiovascular system depression. Very few reports of human intoxication have been documented so far.

Keywords: Amitraz poisoning, pesticide poisoning, human intoxication

INTRODUCTION

Amitraz is a pesticide used to control ectoparasites in canines, tics and mites in cattle and sheep, psylla infection in pears and red spider mites in fruit crops[1,2]. It consist of triazapentadiene (1,5di-(2,4-dimethylphenyl)-3-methyl-1,3,5-tri-azapenta-1,4diene)[3], an insecticide from the formamadine family. Commercially available preparations contain about 12.5-20% of the compound in organic solvents[4].

Amitraz poisoning is uncommon in humans. It occurs via oral, dermal or inhalational routes[1,2,5,6,7]. The most common route is oral route[8,9]. Most of the human intoxications are of accidental ingestion by children[9-14].

The clinical manifestations are central nervous system depression (drowsiness, convulsions and coma), miosis or mydriasis, respiratory depression, cardiovascular depression (bradycardia, hypotension), vomiting and decreased gastrointestinal motility. These manifestations are because of the alpha adrenergic agonist action of amitraz. No specific antidote has been tried yet on humans, leaving supportive management the only treatment.

CASE HISTORY

A 22 year old female presented following consumption of an unknown poisonous substance in the casualty. As per history given by her husband, she had consumed about 20-25 ml of a liquid chemical named “amitraz” about 30 minutes back. She then started having episodes of vomiting and turned drowsy.

On examination her GCS was 11/15, pupils were bilaterally dilated. She had a heart rate of 68 beats per minute, blood pressure of 110/70 mmHg, respiratory rate of 14 per minute, SPO2 of 94%. Gastric lavage was performed, intravenous fluids were given, injection ranitidine and injection perinorm were given stat and patient was put on oxygen inhalation with venturi mask FiO2 60%, flows @12litre per minute.

At about 2:00 am in the night, her heart rate dropped to 36 beats per minute following which injection atropine 0.6mg iv stat was given. Infusion atropine @ 1ml/hr was then started.

The next day, patient started having respiratory distress. On examination, her

heart rate was 74 beats per minute, blood pressure-90/60mmHg, respiratory rate-30/minute, SPO2- 70%, GCS-9/15, pupils-bilaterally dilated. On chest examination, bilateral coarse crepts were heard. Cardiovascular examination was normal. Arterial blood revealed respiratory alkalosis with pH 7.47, pCO2 of 30 mmHg, HCO3 of 21.6 mmol/L, pO2 of 65 mmHg. The patient was intubated, shifted to ICU and put on a ventilator on SIMV mode, following which saturation improved to 100%

Complete blood count, serum urea, creatinine, electrolytes, liver function tests, urine routine and microscopy examination were normal.

After two days the patient was extubated. Her vitals were as following: GCS-15/15, heart rate- 70 beats per minute, BP-116/84mmHg, SPO2- 94% with O2@5 litres/minute, respiratory rate- 16/minute. The patient was given nebulisation with duolin and budecort respules.

The same night at about 12:30 am, the patient started complaining of uneasiness. On examination: Heart rate was irregular 85-120 beats/minute, BP-94/66 mm Hg. On ECG monitor multiform PVC's were seen. On CVS examination- normal heart sounds were heard. Injection loxicard 100mg IV stat was given and simultaneously cardiology call was made and bed side echocardiography done in which no gross abnormality was detected. Tablet ranolazine 500mg BD and tablet metoprolol 6.25mg QID were started. Subsequently, the ECG changes subsided. There was no further complaint from the patient. She recovered fully within 48 hours and was shifted from ICU to the female ward and was later discharged on day 7.

DISCUSSION

Amitraz is an alpha2 adrenergic receptor agonist. It stimulates alpha2 receptors in the CNS, alpha2 and alpha1 receptors in the periphery[15] and also inhibits monoamine oxidase enzyme activity and prostaglandin E2 synthesis[16,17]. Organophosphate or carbamate toxicity also share similar clinical features[18]. The minimum toxic dose reported by Jorens P.G. et al. is 3.57mg/kg[3]. In our case report, patient had consumed 2500mg of amitraz orally (41.6 mg/kg). The clinical features include CNS depression, respiratory depression and cardiovascular effects.

The reported onset of action in most studies is between 30-180 minutes following ingestion[1,6]. In a study by Yaramis, A. et al., CNS depression was observed within 30-90 minutes[10]. Aydin, K. et al., in his study reported CNS depression within 30-120 minutes[7]. Kalyoncu[8] had reported 5 minutes to 6 hours onset of action for oral route and 5 minutes to 24 hours for dermal exposure. In our case report, the patient turned drowsy 30 minutes after amitraz ingestion.

In our case, drowsiness was a significant manifestation[19,20]. The GCS first dropped to 11/15 and further dropped to 9/15. It was due to the alpha 2 agonist action of amitraz. Yilmaz, H. L., in his study documented disorientation, drowsiness and a GCS scale of 9/15 as a clinical manifestation of amitraz poisoning[1]. Ertekin, V. et al., reported generalized seizures following amitraz poisoning[6]. Coma[19] and vomiting[21] were also described. Shitole, D. G. et al., reported cerebral edema in the CT brain of a patient following amitraz poisoning[21].

Miosis with absence of light reflex is commonly seen[22]. Mydriasis is less commonly described. However in our case report, patient had bilaterally dilated pupils. This is because at low doses, alpha 2 adrenergic agonists induce miosis by its effect on presynaptic receptors and in higher doses causes mydriasis by its action on postsynaptic receptors

Amitraz poisoning causes bradycardia and hypotension via its alpha 1 and alpha 2 agonistic action which was seen in many case reports. Aydin, K. et al., in his study reported non specific ST changes in the ECG[12]. In our patient, bradycardia and premature ventricular contractions were

reported which were treated with injection atropine and loxicard respectively.

Respiratory depression requiring mechanical ventilation has been recorded in some cases[11,19]. Kalyoncu et al reported respiratory alkalosis in two cases, respiratory acidosis in three cases and metabolic acidosis in five cases[8]. In our patient also respiratory depression was observed which required intubation. Arterial blood gas analysis revealed hypoxia with respiratory alkalosis.

The renal function tests, serum electrolytes, liver function tests were found to be normal in our case. However hypernatremia has been rarely reported[8,19]. Ertekin et al had reported elevated alkaline phosphate levels in his study[6].

There is no specific antidote for amitraz poisoning and treatment remains mainly symptomatic management. In many studies, gastric lavage and activated charcoal have been tried. Yilmaz et al in their study recommended gastric lavage to be performed only in massive doses. Atropine sulphate has been used in patients who develop bradycardia. Yilmaz H.L. concluded that atropine is effective only in symptomatic bradycardia.

Several alpha2 adrenergic antagonists have been tried in animals to reverse the effects of amitraz. Yohimbine, alpha2 antagonist prevented amitraz induced hyperglycemia, CNS depression, gastrointestinal effects, bradycardia, mydriasis. Atipamezole, an alpha2 adrenergic antagonist also reversed the effects of amitraz with less side effects compared to yohimbine.

In our patient, the management was mainly supportive and symptomatic. Gastric lavage was performed initially. Bradycardia was treated with infusion atropine. For respiratory depression, patient was put on mechanical ventilation. The premature ventricular contractions on ECG subsided with loxicard. With the symptomatic management, the patient improved significantly.

CONCLUSION

In our study, we presented a young female patient with 41.6mg/kg of amitraz poisoning who developed CNS depression, bradycardia, and respiratory depression requiring mechanical ventilation. These clinical manifestations can be explained by the agonist action of amitraz on alpha1 and alpha2 receptors. Management is considered to be symptomatic and supportive. Gastric lavage is to be done during early hours of ingestion. Atropine is the first line of therapy for bradycardia. Prognosis is good and patient may be discharged healthy without any organ dysfunction.

DECLARATIONS

Declaration of Patient Consent

Appropriate patient consent was taken prior to publication in the journal.

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Conflict of Interest: None

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