

CLINICAL PRESENTATION – REPORT ON AN UNUSUAL POISONING IN HUMAN



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Abstract

Unusual suicidal poisoning is associated with ingestion of rare household poisons, because as the casualty would grasp any non edible dangerous chemical substance available in the eyes of the victim. We have managed a 38 yr old woman a case of suicidal intake of amitraz, a non-systemic acaricide, an ectoparasite repellent and insecticide used in veterinary medicine. Amitraz intake is rarely lethal and management is normally symptomatic.

This female was brought to the hospital within few hours of ingestion of Amitraz 30ml (1 bottle) at 10.30pm at her home, immediately she has been taken to nearby clinic and given first aid with inj. Atropine 4 ampules through IV, charcoal administered through ryles tube to induce vomiting and started with Ringer lactate 300ml as IV infusion. As the patient did not show any response she was brought to Apollo Specialty Hospital. On physical assessment the patient had extreme dryness of the airway, spontaneous breathing with the RR 28 br/mt, low peripheral oxygen saturation of 65%, heart rate 110 b/m, blood pressure is 100/60 mmHg. She was irritable, not responding to comments. The ABG analysis showed severe respiratory acidosis and metabolic acidosis. She was initiated with symptomatic treatment, there was a slow prognosis in the hemodynamic status after 12 hours of treatment and discharged stably after 36 hours.

Key words: Amitraz poisoning, Acaricide, pesticide, metabolic and respiratory acidosis, symptomatic treatment.

Introduction

Amitraz is a triazapentadiene compound, a member of the amidine chemical family. It is the

insecticide/acaricide for controlling the ectoparasites in animals. Commercial formulations of amitraz generally contain 12.5-20% of the drug in organic solvents, especially Xylene, which is also used as a solvent in paints, cleaners, and glues. A limited number of human intoxication cases have been reported in the reviews which are distributed worldwide particularly in Southeast Asia. This pesticide poisoning commonly consumed through oral, inhalational (most potential), and dermal routes. The toxic effects of amitraz are due to its α_2 -adrenergic agonist actions in the central nervous system and both α_1 and α_2 adrenergic receptor stimulation in the periphery. It also inhibits monoamine oxidase (MAO) enzyme activity and prostaglandin E2 synthesis, though some of these effects may be dose dependent.

Goal

The main focus of this case scenario presentation is to converse the management of rare poisoning among nurses because of lack of a clear and specific protocol for Amitraz poisoning this presentation provides an useful and valuable scientific information for health care professional to manage the patient confidentially.

Mechanism of Action

The acaricidal and insecticidal activity of amitraz is due to its antagonistic effect on octopamine receptors of the nerve cells in the brain. Parasites become hyperexcited, paralyzed and eventually die.

Toxic symptoms caused by Amitraz poisoning

- Hyperglycemia. (excessive sugar in the blood)

- Hypothermia. (too low body temperature)
- Polyuria. (excessive urination)

Gastrointestinal symptoms

- Lack of appetite.
- Nausea and vomiting.
- Abdominal pain.
- Reduced gut motility.

Central Nervous System (CNS)

- Sedation. (reduced irritability or agitation)
- Depression.
- Tremor. (uncoordinated trembling or shaking movements)
- Hypotension. (low blood pressure)
- Bradycardia. (too low heart rate) that can turn to tachycardia (too high heart rate) and hyperventilation (too rapid breathing).

Antidote and treatment of Amitraz intoxication. (Human and Animals)

1. Atipamezole, 50 mcg/kg IM. The effect is very fast but lasts only 2-4 hours. After this first treatment it may be necessary to administer Yohimbine (0.1 mg/kg p.o.) every 6 hours till complete recovery.
2. Yohimbine 0.1 mg/kg IV is usually the best option. It displaces amitraz from the alpha 2 receptors, which relieves bradycardia, hypotension, and sedation, and restores gut motility.

Clinical case presentation

Mrs. Revathi, 38 year's female consumed amitraz pesticide 30ml (1 bottle) at 10.30pm at her home. Immediately she was taken to a nearby clinic and was administered inj. Atropin, 4 ampules through IV. Stomach wash given with 300 ml of charcoal. Ringer Lactate was started as Intra venous bolus. After all the first aid measures there the patient maintained very poor oxygen saturation. So she was immediately shifted to Apollo Speciality Hospital, Madurai.

On examination there were no secretions in the airway, spontaneous breathing, but poor saturation.

Her oxygen saturation was 65%, respiratory rate was 28 br/m, heart rate 110 bts/m, blood pressure 100/60mmHg, and she was irritable, not obeying commands. ABG analysis revealed severe respiratory and metabolic acidosis. On receiving the patient in the emergency inj. Pan 40mg IV and inj. Emeset 8mg, inj. Ketamine 50 mg IV, inj. Fentanyl 50 mg IV was administered and intubated orally with seven size endotracheal tube. Then activated charcoal 60 mg and sorbitrate syrup was given through ryles tube. The patient was started with inotrops dopamine 10mcg/hr, noradrenalin 20mg/hr and plasmolyte A 100ml/hr.

Day 1:

She was on controlled mode ventilator FiO2 - 60%, Tv- 45, PEEP- 5, rate- 15, GCS: eye opening - 3, verbal - she was on ET tube (unable to assess), Motor response - 4, total score is 7/15. Pupils - 2.5 mm dilated bilaterally, Investigation results showed random blood sugar - 245 mg/dl, Sr. Cholinesterase - 9727, HbA1C - 6.3, Hb - 12.8gms%, WBC - 19,300 cmm, RBC - 4.7, Sr. Urea - 35, sr. Creatinine- 0.4, Uric acid - 4.0, magnesium - 2.1mg/ dl, sodium- 137 mEq/ L, pottassium - 4.0 mEq/ L, Chloride - 102 mEq/ L. ABG analysis revealed PCo2 - 66.6 mmHg, PO2- 47.9mmHg, HCO3 - 23.0mmol/L, the patient was on Inj. Human actrapid Q6H on sliding scale, Inj. Fentanyl 10mcg IV, Inj. Nexium 40mg IV bd, Inj. Lesuride 25mg IV bd, Inj. Magnex forte 1.5gm IV bd, smoisol 2 drops instillation Tds, Activated charcoal 30mg through ryles tube Tds

Day 2:

Patient was on spontaneous mode ventilation and planned for extubation. The GCS score improved to 10/ 15. Central line is in right jugular vein. The patient was started with Neb. Ipravent 2cc Q6H, neb. Budecort 2cc Q6H, vitals are stable - Heart rate - 110 b/m, respiratory rate - 16b/m, B.P 110/60mmHg, SPO2 is 98%. The investigation was at the normal limits. She is conscious and obeying commands. Her braden scale was below 11/23.

Day 3:

She was conscious and oriented to time place and person, extubated, ryles tube removed, activated

charcoal stopped, inj. Dopamine infusion was stopped. IVF NS 100ml/ hr on flow and later reduced to 70ml/hr. Now she is on multi channel monitor, her B.P is 110/70 mm Hg, liquid diet advised and eye drops moisol was stopped. Urine output was 10 ml/ hr and inj. Lasix 10 mg IV was given, 5 L of oxygen was on flow, no other specific complaints. She was shifted to the ward.

Day 4 – 7

The patient was hemodynamically stable from day 4. She was on solid diet from day 5 onwards. Bowel and bladder pattern was regular from day 6. Eyes reacting to light and no dryness of eyes noticed. All the drugs were stopped from day 6. She was discharged on day 7 of hospitalization.

Discussion

Amitraz shows toxic effects on both humans and animals. This type of poisoning is commonly noted in animals but rare among humans. Amitraz is slightly toxic. Amitraz shows hepatotoxic, CNS stimulative or depressive effects. It can cause gastric stasis. Amitraz leads to rise in plasma glucose level and suppress insulin release. Decreased body temperature is due to inhibitory effect of Amitraz on Prostaglandin E2 synthesis. Even after poisoning by potentially lethal dose of Amitraz, studies have shown complete recovery. As there is no specific antidote for Amitraz Poisoning the medical management with O2 supplementation, airway maintenance, hydration and supportive management are the key dimensions for complete recovery of the patient. As in recent days many pesticides to create an awareness about Amitraz poisoning and must be created among farmers and their family members also. Public Health Education and instructions to drug producing companies will be necessary to decrease the incidence of Amitraz Poisoning.

Outcome

In spite of a rapidly progressing and life-threatening clinical picture, amitraz intoxication in humans carries a low morbidity and mortality when appropriate supportive treatment is given. Regarding Ms.Revathy though she had severe toxic effects of the drug during the first 3 to 4 days, after starting the

appropriate supportive treatment and management, her recovery was very fast.

Nursing Diagnosis

- Ineffective airway clearance related to respiratory stimulative effect of pesticide.
- Ineffective airway clearance related to excessive secretion.
- Impaired tissue perfusion (cerebral) related to blocking of nerve receptors.
- Imbalanced nutrition less than body requirement related to drowsiness.
- Knowledge deficient related to lack of awareness regarding amitraz poisoning.

Nursing Management

- Assessed the patient's ACB, vitals and consciousness every two hourly.
- Assisted in intubation and suction as needed to keep the airway clear.
- Monitored and recorded hourly BP as the patients were on inotropha.
- Adminstered IV fluid to maintain the hydration status. Maintained I /O chart daily.
- Instill eye drops every two hourly to prevent dryness of mouth.
- Administered antibiotics to prevent respiratory infections.
- Health education was given about the pesticide poisoning.

Conclusion

To conclude the basic care pathway of a patient with amitraz poisoning focuses on initial stabilization, reducing absorption, and increasing elimination of the toxin. Despite a life threatening clinical picture, amitraz poisoning in humans carries a low mortality when appropriate supportive therapy must be given immediately. Recovery usually occurs within 12-48 hours and the patients are discharged without any organ dysfunction.

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Someone is sitting in the shade today
because someone planted a tree a long time ago.

-Warren Buffett

Adolescents with Nocturnal Enuresis and Daytime Urinary Incontinence

A study published in the *Neurourology and Urodynamics* reviewed studies on prevalence, clinical symptoms, and associated risk factors, as well as formulated recommendations for the assessment and treatment of nocturnal enuresis (NE) and daytime urinary incontinence (DUI). It was found that 1-2% of older adolescents are affected by NE and 1% by DUI. Moreover, NE and DUI are associated with multiple risk factors, such as fecal incontinence and constipation, obesity, chronic illness, and psychological impairment. Adolescent NE and DUI can be treated by a multidisciplinary team based on pediatric principles. It was also stated that additional treatment components have been developed for adolescents.

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