



Amitraz Poisoning in a Patient from Rural Africa

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Abstract:

Amitraz is a pharmacologically active chemical compound widely used in Africa as an ectoparasiticide and agricultural pesticide. This article reports a 25 year old female from rural Kenya who presented to emergency department with fourteen hours history of unconsciousness and mydriasis after having ingested an unknown poison. She was erroneously treated as a case of organophosphate poisoning but later managed supportively with intravenous fluids, airway management and monitoring of urine output. The importance of considering chemicals other than organophosphates in patients with poisoning as well as the supportive management of patients with amitraz intoxication is highlighted. Public education on potential hazards of amitraz poisoning is of vital importance for community health.

Keywords: Amitraz toxicity, Organophosphate toxicity, Poisoning

Introduction

Amitraz is a chemical belonging to the formamidine class of compounds. It is an acaricide used to control ticks in cattle, sheep and dogs and as an agricultural pesticide. It is available as 12.5% or 25% (m/v) concentrated emulsion formulations with xylene (an aromatic hydrocarbon) used as a solvent [1]. In Kenya and most of Africa, it is mainly marketed as Triatix®.

Amitraz is a pharmacologically active compound that acts on the central nervous system (CNS) by activating pre-synaptic alpha-2 adrenoceptors to cause inhibition of noradrenaline release resulting in suppressed outflow of sympathetic nervous activity from the CNS [1]. This may cause unconsciousness, bradycardia, miosis, mydriasis, hypotension, hyperglycaemia, glycosuria, vomiting and respiratory failure [2-3].

Poisoning by amitraz in humans occurs either through ingestion of the chemical or through contact with skin. It occurs mostly in children and is often accidental [4-5]. The presence of bradycardia combined with miosis can point towards mistaken diagnosis of organophosphate poisoning and inappropriate treatment in up to 17.4% of cases [1, 6]. Management is mainly supportive and mortality is low if care is appropriately administered [7].

Case Report

A 25-year-old African woman presented to the Busia district hospital (BDH) in Western Kenya approximately 14 hours after having ingested an unknown amount of poison following a domestic dispute. She was initially taken to a health centre near her home where no intervention was attempted and was later referred to BDH for further management. Examination revealed an unconscious woman with a Glasgow Coma Scale (GCS) score of 3/15. Her pupils were bilaterally dilated and with a slow reaction to light. Vital signs on admission were: blood pressure 100/69 mm Hg, pulse rate of 80 beats per minute, temperature of 36.8°C and a respiratory rate of 15 cycles per minute. The rest of the examination was unremarkable.

Organophosphate poisoning was assumed and she was started on intravenous (i.v) atropine, 10 mg in 500 ml normal saline to run in 2.5 hours. She also received one dose of 40 mg i.v furosemide. A nasogastric tube was fixed and gastric lavage done. Later, she was put on maintenance intravenous fluids, catheterised and an input/output charting started. Laboratory studies done included a complete blood count, liver function tests, serum creatinine levels and a urinalysis all of which were unremarkable.

The following day, a close relative revealed that she had ingested Triatix® (amitraz) and the diagnosis of organophosphate poisoning was dropped. Her GCS score had improved to 7/15 but her pupils were still dilated with a sluggish reaction to light.

There was no neck stiffness and no focal neurological deficits. Urine output was normal and supportive management was continued. On the third day of admission, she regained consciousness with a GCS score of 14/15. However, she was noted to be disoriented and hallucinating. There was dilatation of the pupils with slow reaction to light. Since she was able to swallow, the nasogastric tube was removed. She was still weak and could not ambulate.

On day four of admission, she was able to sit and eat alone but was still disoriented and hallucinating. Her pupils were now normal in size and adequately reacting to light. By the fifth day of admission she had become ambulant but with a slightly ataxic gait and was oriented with improved cognitive function. Her urinary catheter was removed and by the sixth day of admission she was ready for discharge after a full psychiatric assessment.

Discussion

Amitraz poisoning is under reported in Africa despite its widespread use as an ectoparasiticide and agricultural pesticide. Only one study, from the Republic of South Africa, describes this phenomenon in Africa [1]. The presentation of CNS depression, mydriasis and normal laboratory findings in this case of amitraz poisoning resembles presentation described elsewhere in the literature [1,6,8,9]. Consciousness may be regained in 6 to 22 hours in both children and adults [4,6,10-12]. However, in this patient, the time to regaining consciousness in 38 hours (from ingestion of poison) is longer than that reported in adults from Turkey and South Africa [1,11] and is probably due to a delay in seeking medical help (the patient presented 14 hours after ingesting amitraz). However, this observation may also be due to a high dose of ingested poison.

The presence of disorientation, hallucinations and an ataxic gait in this patient is an unreported feature of amitraz poisoning. However, xylene (a hydrocarbon solvent used to dilute amitraz) is known to cause neuroexcitation, altered mental status, motor in-coordination and ataxia [13,14] and may be responsible for this presentation. The suicidal intent of this patient is consistent with that reported in other cases from Africa [1].

Clinical presentation in patients with amitraz poisoning includes CNS depression, respiratory failure, bradycardia, myosis, mydriasis, hypothermia, hypotension and vomiting [4,5,7,10,11,15,16]. These are the result of stimulation of alpha-2 adrenoceptors by the poison. The effect of amitraz on CNS depression and pupil size is thought to be dose dependent. In a retrospective study of 44 adults, Demirel et al [9] showed a correlation between the amount of amitraz ingested and the length of CNS depression observed. Atabek and colleagues suggest that higher doses of amitraz results in mydriasis while lower doses leads to myosis [10]. Amitraz is also known to inhibit the enzyme monoamine oxidase and to inhibit prostaglandin synthesis but the clinical significance of these actions remains unknown [10].

Laboratory findings in amitraz intoxication include hyperglycemia, glycosuria and elevated transaminase levels (aspartate and alanine aminotransferases) [2,9,11,16]. Amitraz is thought to reduce insulin secretion which contributes to hyperglycemia. In rats, it has been observed to decrease hepatic glutathione activity [2] but the mechanism of elevated liver function tests in man is not known [10]. Renal function has been observed to remain unaffected but there are reports of polyuria in both adults and children [8,9,11].

Since there is no known antidote for amitraz intoxication, management is mainly supportive and mortality is low if care is appropriately administered [6,7]. The main approach involves hemodynamic stabilization with intravenous fluids, airway management and oxygen if needed [12]. Gastric lavage may be done and activated charcoal given. Emetics are contraindicated because of the increased risk of aspiration [10]. Caution should be taken not to mistake amitraz poisoning for organophosphate poisoning as up to 17.4% of cases are misdiagnosed [1]. In conclusion, this case report highlights the reversible nature of amitraz intoxication as well the importance of adequate supportive management. Clinicians should be sensitized to the fact that organophosphate poisoning is an important cause of poisoning in rural Africa, it is not the only cause of poisoning and other causes for the patient's presentation should be sought.

Acknowledgment

The author acknowledges the care and attention granted to the patient by her caretakers and all the staff of the female medical ward in Busia District Hospital.

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